

monitored flights were consistent with acceptable levels for discouraging the survival and growth of microorganisms. Cargo compartments in aircraft should be kept free of animal excrement and arthropods. Pathogenic microorganisms should not be transported on aircraft carrying passengers (National Research Council, 1986).

This study does not address the role of viruses as infectious agents in the cabin air environment. The relative importance of viruses as sources of indoor-related illnesses (e.g., influenza) can be seasonally related (Joklik 1985). Additionally, in the case of influenza viruses, the periodicity of epidemics and pandemics is related to the genetic stability of the virus and the appearance of a new virus with altered surface antigens (Joklik 1985). The monitoring conducted for this investigation occurred during the spring/summer season and not the winter season, which is associated with an increase in virus-related illnesses (Joklik 1985). Monitoring of viruses in aircraft cabins was not undertaken because of contractual constraints. To meet the contract schedule, monitoring had to be conducted during April through June when seasonal prevalence of viruses would have been low. Thus no monitoring for viruses was conducted. Nonetheless, viruses are recognized as the predominant etiologic agent for respiratory infections, estimated to cause 50 to 60 percent of all community-acquired illnesses (Feeley 1985).

8.2 COSMIC RADIATION

8.2.1 Exposure to Cosmic Radiation

The major source of radiation exposure to humans is natural in origin. This includes external sources such as cosmic radiation and terrestrial radiation from radioactive substances in the ground and building materials, and internal sources such as naturally occurring radionuclides in the body inhaled or ingested from air and diet. Natural radiation exposes virtually the world population at a relatively constant rate throughout time and is virtually independent of human activity.

According to the United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR), the mean annual effective dose equivalent is estimated to be 2.4 millisieverts (mSv) per year or 240 millirem (mr) per year¹ (UNSCEAR, 1988).

For airline passengers and cabin crew members, the major contributing factor to any increase in the overall radiation dose is cosmic radiation, high energy radiation that enters the atmosphere from cosmic space originating usually at either the sun or in deep space. Primary cosmic rays enter the atmosphere and interact with the nuclei of atoms present in the air, resulting in the formation of secondary cosmic rays such as neutrons, protons, pions, and kaons, and a variety of reaction products (cosmogonic nuclides) such as ^3H , ^7Be , ^{10}Be , ^{14}C , ^{22}Na , and ^{24}Na . The high-energy secondary cosmic rays thus formed react further with nuclei in the air to form additional secondary particles (electrons and muons).

In the lower atmosphere, dose rates from the ionizing component vary little with latitude but significantly with altitude, doubling approximately every 1,500 meters (4,875 feet). Measures of absorbed dose rates in air, derived from ionization chamber measurements on aircraft, yield dose rates of 30 nGy/hr (one Gray, Gy, is equal to 100 RAD) at sea level for any latitude and from there increase to about 4 $\mu\text{Gy/hr}$ at an altitude of 12 km (39,600 feet) closer to the poles. At sea level, the absorbed dose rate in outdoor air from the ionizing component of cosmic rays was reported to be 32 nGy/hr (UNSCEAR 1982). This value was taken to be numerically equivalent to the effective dose equivalent. The doses are somewhat lower indoors due to the shielding effect of building structures. A shielding factor of 0.8 has been used to yield an average indoor absorbed dose index rate (at sea level) of 26 nGy/hr (UNSCEAR 1988). Using a value of 1 for the quality factor and an indoor occupancy factor of 0.8, the annual effective dose equivalent is estimated to be about 240 μSv per year at sea level.

¹ 1 millisievert is equal to 100 millirem.

Variation of the neutron component with altitude and latitude is similar to that of the ionizing component. At sea level, the neutron flux rate is approximately $0.008 \text{ cm}^{-2}\text{s}^{-1}$. Using an estimate of 2.4 nSv/hr for the average dose equivalent rate, and neglecting the shielding effect of building structures, the annual effective dose equivalent for the neutron component is estimated to be about 20 uSv at sea level (UNSCEAR 1982).

When the data are transformed to a cumulative effective dose equivalent as a function of altitude, a per capita effective dose equivalent for the world population is found to be 355 uSv (not time dependent), with the ionizing component accounting for 300 uSv and the neutron component accounting for 55 uSv . This increased dose equivalent estimate is due to the range of altitudes and latitudes in which people live. It is important to note that the dose equivalent from the neutron component, which is small at sea level, increases more rapidly than the dose from the ionizing component and becomes more important at altitudes above 6 km ($19,800 \text{ feet}$).

Elevated exposures result from prolonged presence at high altitudes. Populations living in such high altitude cities as Bogota, Lhasa, or Quito receive annual effective dose equivalents from cosmic radiation in excess of 1 mSv . It follows that commercial airliner passengers and cabin crew members will be exposed to higher dose rates than the general nonflying population. These dose rates will vary according to flight altitude, flight latitude, and the amount of solar activity.

With decreasing altitude from the top of the atmosphere, the dose equivalent rate from galactic radiation first increases, then decreases. The increase is a consequence of the multiplicity and characteristics of the secondary particles produced after collision of high energy cosmic particles with the atomic nuclei of gases in the atmosphere. Many of the impacting and generated particles maintain enough energy to form additional secondary particles. The altitude at which the dose equivalent rate is maximum depends on the geomagnetic latitude. With decreasing altitude below 21.2 km ($70,000 \text{ feet}$) at all latitudes, continued energy

degradation and cannibalization of particles results in a decreasing dose equivalent rate. In the contiguous United States, the dose equivalent rate at 12.1 km (40,000 feet) is about 40 percent of the rate at 21.2 km (70,000 feet) (Federal Aviation Administration 1989).

The geomagnetic field of the earth deflects many charged particles of solar and galactic origin that would otherwise enter the atmosphere. Shielding is most effective at the geomagnetic equator, where the geomagnetic lines of force are nearly perpendicular to the surface of the earth. At airliner cruise altitudes, the cosmic radiation dose equivalent rate over the geomagnetic poles is approximately twice that over the geomagnetic equator. Most high-altitude flights of U.S. commercial aircraft occur with scheduled flights between the United States and Europe or Asia (Federal Aviation Administration 1989).

The cycle of rise and decline in the intensity of the cosmic radiation incident on the atmosphere lasts approximately 11 years, with the intensity inversely related to solar activity. Charged particles are continuously ejected from the sun but are generally too low to contribute to the radiation level at airliner flight altitudes. On infrequent occasions, the energy levels and quantities of ejected solar particles are high enough to substantially increase the dose equivalent rate at typical cruise altitudes. During the period from 1956 to 1972, there were four solar particle events during which the dose equivalent rate on polar routes at 12.4 km (41,000 feet) probably exceeded 100 mSv/hr (Federal Aviation Administration 1989)

Dose equivalents for flights typical of continental U.S. latitudes and circumpolar transoceanic routes are presented in Table 8-4. Since total radiation dose is the simple sum of individual exposures, this table enables any individual to ascertain cumulative radiation dose by adding appropriate flights (as actually listed or as representatives of similar flights) according to their specific frequencies of occurrence. The summed value represents the relevant individual exposure in the determination of risk, as described in Section 8.2.3.

TABLE 3-4. DOSE EQUIVALENTS FROM GALACTIC COSMIC RADIATION RECEIVED ON AIRLINER FLIGHTS

Origin - Destination	Single Nonstop One-way Flight			
	Highest Altitude KM (feet, thousands)	Air Time (in hrs)	Block Time ¹ (in hrs)	Dose ² (in microsieverts)
Houston - Austin	6.1 (20)	0.5	0.6	0.1
Seattle - Portland	6.4 (21)	0.4	0.6	0.1
Miami - Tampa	7.3 (24)	0.6	0.9	0.4
St. Louis - Tulsa	10.7 (35)	0.9	1.1	2.0
Tampa - St. Louis	9.4 (31)	2.0	2.2	5.4
San Juan, PR - Miami	10.7 (35)	2.2	2.5	7.2
New Orleans - San Antonio	11.9 (39)	1.2	1.4	4.3
Denver - Minneapolis	10.1 (33)	1.2	1.5	4.7
New York - San Juan, PR	11.3 (37)	3.0	3.5	13.0
Los Angeles - Honolulu	10.7 (35)	5.2	5.6	22.0
Chicago - New York	11.3 (37)	1.6	2.0	8.5
Honolulu - Los Angeles	12.2 (40)	5.1	5.6	25.0
Washington, DC - Los Angeles	10.7 (35)	4.7	5.0	24.0
Tokyo, Japan - Los Angeles	11.3 (37)	8.8	9.2	48.0
Los Angeles - Tokyo, Japan	12.2 (40)	11.7	12.0	62.0
New York - Chicago	11.9 (39)	1.8	2.3	12.0
Minneapolis - New York	11.3 (37)	1.8	2.1	11.0
London - Dallas/Ft. Worth	11.9 (39)	9.7	10.1	53.0
Dallas/Ft. Worth - London	11.3 (37)	8.5	8.8	49.0
Seattle - Anchorage	10.7 (35)	3.4	3.7	21.0
Lisbon - New York	11.9 (39)	6.5	6.9	41.0
Chicago - San Francisco	11.9 (39)	3.8	4.1	26.0
Seattle - Washington, DC	11.3 (37)	4.1	4.4	29.0
London - New York	11.3 (37)	6.8	7.3	49.0
New York - Seattle	11.9 (39)	4.9	5.3	36.0
San Francisco - Chicago	12.5 (41)	3.8	4.1	29.0
Tokyo - New York	12.5 (41)	12.2	12.6	91.0
London - Los Angeles	11.9 (39)	10.5	11.0	80.0
Chicago - London	11.3 (37)	7.3	7.7	56.0
New York - Tokyo, Japan	13.1 (43)	13.0	13.4	99.0
London - Chicago	11.9 (39)	7.8	8.3	62.0
Athens, Greece - New York	12.5 (41)	9.4	9.7	93.0

¹The block hours of a flight begin when the aircraft leaves the blocks before takeoff and end when it reaches the blocks after landing.

²For each flight, estimates of dose-equivalent were made using one flight plan, taking into account changes in altitude and geomagnetic latitude from takeoff to touchdown.



8.2.2 Health Effects from Exposure to Cosmic Radiation

There are two types of effects from exposure to radiation: nonstochastic and stochastic. Nonstochastic effects are those for which the probability and severity of the effect vary with dose and a threshold for the effect exists. Examples of nonstochastic effects include pancytopenia following irradiation of bone marrow, and pneumonitis and pulmonary fibrosis following irradiation of the lung. Stochastic effects are those for which the probability of the occurrence of effect, and not its severity, varies as a function of dose in the absence of a threshold. The major stochastic effects are heritable genetic effects and cancer.

Early to intermediate effects of exposure to radiation can be taken to include the somatic effects of exposure to irradiation, excluding carcinogenesis and shortening of life span which are late somatic effects. Genetic effects of irradiation include gene mutation and chromosome aberrations.

Tumors caused by radiation are indistinguishable from tumors caused by other sources (e.g., chemicals), and health effects other than cancer are also very similar to those occurring spontaneously or induced by exposure to other agents. The health effects of radiation are often augmented by other factors that tend to increase overall risk; these include tobacco smoking and dietary factors (UNSCEAR 1988).

8.2.3 Quantitative Estimation of Risk

Risk was determined for cancer, fetal retardation, and birth defects using an algebraic combination of the exposure assessment and dose-response risk coefficients. Radiation risk coefficients used in this investigation were based on UNSCEAR dose-response relationships and modeling protocols (UNSCEAR 1986; 1988). The Fourth Report of the Committee on Biologic Effects of Ionizing Radiation, National Research Council (BIER IV for cosmic radiation) was not complete at the time analyses were conducted.

Risk coefficients for cosmic radiation exposure in utero leading to birth defects, mental retardation, and childhood cancer, as presented

in Table 8-5, were derived from epidemiological studies of children exposed in utero during the bombing of Hiroshima and Nagasaki. The risk coefficient for childhood cancer was assumed to be constant during prenatal development, although there is evidence suggesting that risk is higher in the first trimester (UNSCEAR 1986).

Risk coefficients for adult cancer (solid tumors and leukemia) were derived from epidemiological studies of atom bomb survivors, patients with ankylosing spondylitis (spinal arthritis), and patients with cervical cancer. Estimates were computed using an assumed exposure of 1 Gy and linear dose-response relationship for solid tumors. Additive and multiplicative projection extrapolation models were used to determine risks. Minimum latency for leukemia was set at 2 years and for all other sites at 10 years. The plateau was 40 years for leukemia and lifetime for all other sites. Cancer mortalities in Japan and the United Kingdom were used as baseline mortality rates. The risk coefficients assumed a quality factor of 1. This value is the sum of the relative risk for leukemia and the relative risk for other malignancies (UNSCEAR 1988).

Dose-response plots presented in Figures 8-1, 8-2, and 8-3 for adult cancer, childhood cancer, and fetal retardation and birth defects, respectively, were constructed using the risk coefficients contained in Table 8-5. The procedure for determining risk can be illustrated using the same three example flying profiles presented in Section 7.0 of this report to illustrate cancer risks from exposure to ETS. The parameters of these examples are summarized in Table 7-7. For purposes of illustration, an additional assumption is made here that half of the total flying time indicated for the individuals in the three examples is between New York and Seattle (representing a constant latitude in the continental U.S.) and the other half is between New York and Tokyo (representing a circumpolar flight at high altitude). Additional flights, and their associated cumulative doses and cancer risks, are presented in Tables 8-6 and 8-7 for domestic and international flights, respectively. In each case, flights of varying duration, latitude, and direction were chosen as examples. It

TABLE 8-5. RISK COEFFICIENTS FOR A RANGE OF HEALTH EFFECTS ASSOCIATED WITH EXPOSURE TO COSMIC RADIATION

Health Effect	Risk Coefficient	Period of Vulnerability
Fetal structural abnormalities	500/1 million/mSv	Weeks 2-8 of pregnancy
Mental retardation in fetus	400/1 million/mSv 100/1 million/mSv	Weeks 8-15 of pregnancy Weeks 16-26 of pregnancy
Childhood cancer	20/1 million/mSv	Full term of pregnancy
Adult cancer (leukemia and solid tumors)	4.15/1 million/mSv	

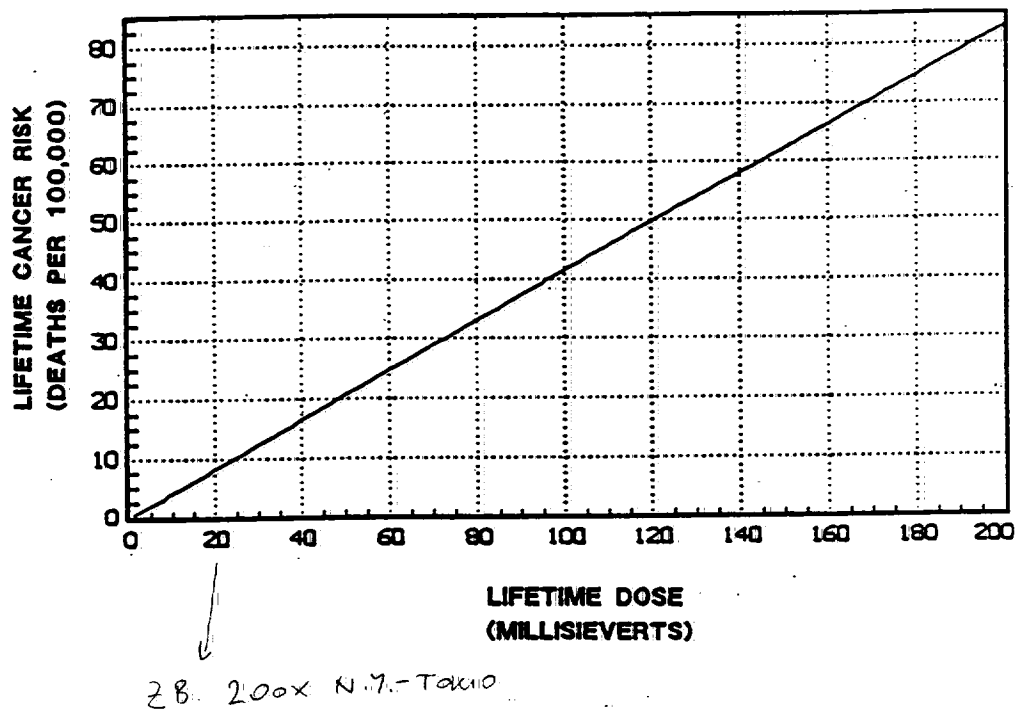


FIGURE 8-1. ADULT CANCER RISK (LEUKEMIA AND SOLID TUMORS) FROM EXPOSURE TO COSMIC RADIATION. ONE MILLISIEVERT = 100 MILLIREM

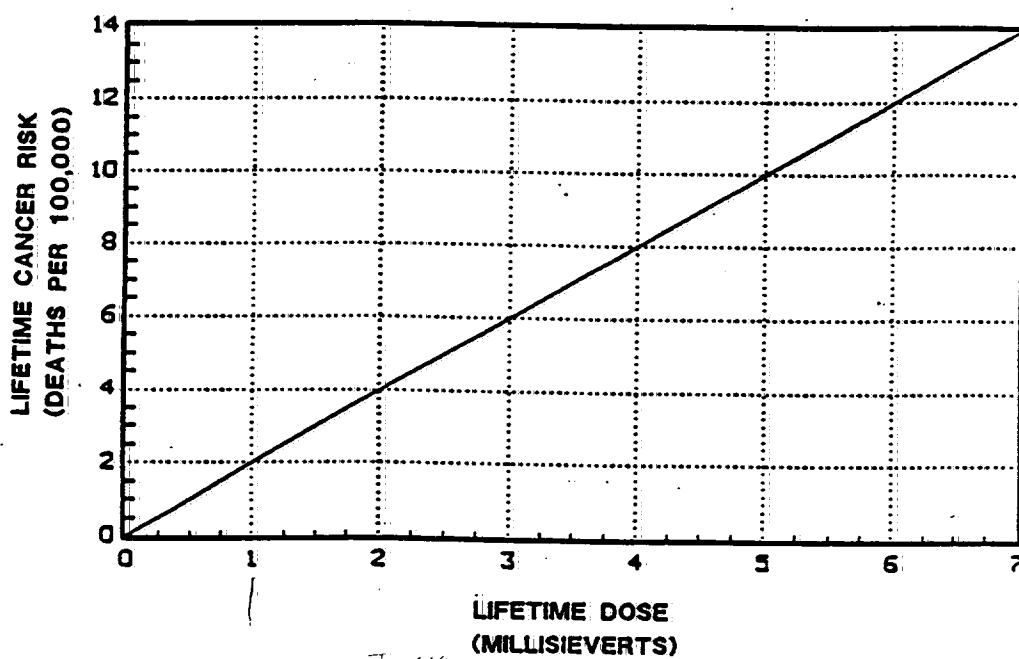
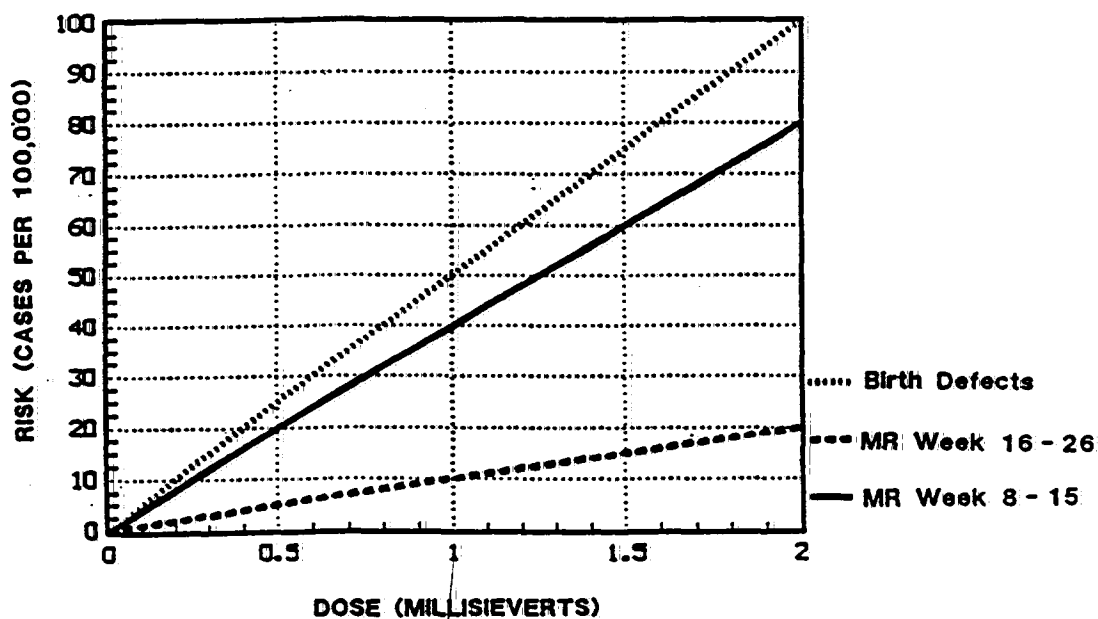


FIGURE 8-2. CANCER RISK PER 100,000 CHILDREN EXPOSED IN UTERO TO COSMIC RADIATION. ONE MILLISIEVERT = 100 MILLIREM



10X N.Y. — Tokyo

FIGURE 8-3. RISK OF MENTAL RETARDATION (MR) AND BIRTH DEFECTS (BD) IN THE FETUS EXPOSED IN UTERO TO COSMIC RADIATION

TABLE 8-6. CUMULATIVE DOSES (20 YEARS) FOR CABIN CREW MEMBERS AND PASSENGERS ON REPRESENTATIVE DOMESTIC FLIGHTS

Representative Flight	Flight Time (Hours)	Dose (μ Sv)	CABIN CREW MEMBERS		PASSENGERS	
			Cumulative Dose Over 20 Years Flying 960 h/yr (mSv)	Cancer Risk (deaths per 100,000)	Cumulative Dose Over 20 Years Flying 480 h/yr (mSv)	Cancer Risk (deaths per 100,000)
Domestic (east/west) New York to Seattle	4.9 ¹	36.0	141.1	59	70.5	29
Domestic (east/west) San Francisco to Chicago	3.8 ²	29.0	146.5	61	73.3	30
Domestic (north/south) Seattle to Anchorage	3.4 ²	21.0	118.6	49	59.3	25
8-26 Domestic (north/south) Tampa to St. Louis	2.0 ³	5.4	51.8	21	25.9	11
Domestic (east/west) Chicago to New York	1.6 ⁴	8.5	102.0	42	51.0	21
Domestic (north/south) Denver to Minneapolis	1.2 ⁴	4.7	75.2	31	37.6	16
Domestic (east/west) St. Louis to Tulsa	0.9 ⁵	2.0	42.7	18	21.3	9
Domestic (north/south) Miami to Tampa	0.6 ⁵	0.4	12.8	5	6.4	3

¹Domestic flights of 4 hours duration or more account for 5.3% of all domestic flights.

²Domestic flights of 3 to 4 hours in duration account for 7.2% of all domestic flights.

³Domestic flights of 2 to 3 hours in duration account for 21.3% of all domestic flights.

⁴Domestic flights of 1 to 2 hours in duration account for 48.7% of all domestic flights.

⁵Domestic flights of 0 to 1 hour in duration account for 17.6% of all domestic flights.

TABLE 8-7. CUMULATIVE DOSES (10 YEARS) FOR CABIN CREW MEMBERS AND PASSENGERS ON REPRESENTATIVE INTERNATIONAL FLIGHTS

Representative Flight	Flight Time (Hours)	Dose (μ Sv)	CABIN CREW MEMBERS		PASSENGERS	
			Cumulative Dose Over 10 Years Flying 960 h/yr (mSv)	Cancer Risk (deaths per 100,000)	Cumulative Dose Over 10 Years Flying 480 h/yr (mSv)	Cancer Risk (deaths per 100,000)
International Circumpolar New York to Tokyo, Japan	13.0 ¹	99.0	73.1	30	36.6	15
International Non-circumpolar Dallas/Fort Worth to London	8.5 ²	49.0	55.3	23	27.7	11
International Non-circumpolar London to New York	6.8 ³	49.0	69.2	29	34.6	14
8-27 International Non-circumpolar New York to San Juan, PR	3.0 ⁴	13.0	41.6	17	20.8	9
International Non-circumpolar San Juan, PR to Miami	2.2 ⁴	7.2	31.4	13	15.7	7

¹International flights of 10 hours duration or more account for 6.5% of all international flights.

²International flights of 8 to 10 hours in duration account for 14.7% of all international flights.

³International flights of 6 to 8 hours in duration account for 14.4% of all international flights.

⁴International flights of 2 to 4 hours in duration account for 34.6% of all international flights.

2028397413

should be noted that the cancer risks for cosmic radiation and ETS are additive.

Example 1. The individual is a cabin crew member who flies 960 hours per year for 20 years. Assuming that 10 years are spent flying from New York to Seattle (dose equivalent of 36 μ Sv for 5.3 hours from Table 8-4), the dose equivalent for this segment is 65 mSv. Assuming that the next 10 years are spent flying from New York to Tokyo (dose equivalent of 99 μ Sv for 13.4 hours from Table 8-4), the dose equivalent for this period is 71 mSv. The total dose equivalent for 20 years of flying is 136 mSv (65 + 71). Referring to Figure 8-1 for adult cancer risk, a lifetime exposure of 136 mSv in flight results in a lifetime cancer risk of 56 cancer deaths per 100,000 or a risk of 1 in 1,786.

Example 2. This individual is a frequent flyer who logs 480 hours per year for 30 years. Assuming that the first 15 years are spent flying from New York to Seattle, the dose equivalent for this period is 49 mSv. Similarly, the dose equivalent for 15 years of flying from New York to Tokyo is 53 mSv. The combined dose equivalent for 30 years of flying is 102 mSv. From Figure 8-1, the risk is 42 cancer deaths per 100,000 or a risk of 1 in 2,381.

Example 3. This individual flies 48 hours/year for 40 years. For the first 20 years, flights between New York and Seattle result in a dose equivalent of 6.5 mSv. For the next 20 years, flights between New York and Tokyo result in a dose equivalent of 7.1 mSv. With a lifetime dose of 13.6 mSv acquired in flight, the risk is 6 cancer deaths per 100,000 or a risk of 1 in 16,667.

Risks for childhood cancer, fetal retardation, and birth defects can be determined in a similar fashion, using the risk coefficients in Table 8-5. For a single transcontinental flight such as Washington to Los Angeles, the dose equivalent is 24 μ Sv. The risks for any of the childhood health effects are very small (less than 1 per 100,000) according to Figures 8-2 and 8-3. For even a high exposure flight such as New York to Tokyo with a dose equivalent of 99 μ Sv, the risks are still small (less than 2 per 100,000).

8.3 OZONE

Ozone levels in airliner cabins were measured on domestic and international flights to determine compliance with current federal stan

dards and to ascertain if observed concentrations pose a health hazard to cabin crew members and passengers.

8.3.1 The FAA Standard for Ozone in Airliner Cabins and its Basis

In 1980, the Federal Aviation Administration (FAA) promulgated an ozone standard for aircraft cabins that included transport category airplanes of commercial air carriers (Federal Register 1980). The standard was prompted by research of the FAA Civil Aeromedical Institute (Federal Aviation Administration 1979, 1980) that demonstrated no significant health effects attributable to ozone at a sea level equivalent of 0.2 ppm for 4 hours, but which did demonstrate respiratory effects in exercising individuals at a sea level equivalent of 0.3 ppm. This suggested a threshold for effect between 0.2 and 0.3 ppm. At a cabin pressure altitude of 1.8 km (6,000 ft), where there is less air for a given volume, 0.3 ppm equates to a sea level equivalent of 0.25 ppm. Accordingly, the FAA established an instantaneous standard of 0.25 ppm (sea level equivalent) and a time-weighted three-hour standard of 0.1 ppm (sea level equivalent).¹

Other regulatory agencies have established similar standards. The Occupational Safety and Health Administration's Threshold Limit Value (TLV)² for the workplace environment is 0.1 ppm. The Environmental Protection Agency's one-hour ambient air standard remains at 0.12 ppm, although recent research on humans under conditions of controlled exposure has suggested the possibility of respiratory effects (i.e., lung infectivity) at ozone levels as low as 0.08 ppm (see below). In addition, there is scientific and regulatory debate over the need for an 8-hour ambient air standard lower than 0.12 ppm. The FAA's standard of 0.1 ppm appears to be in the protective range.

¹ While the actual time-weighted average was 0.08 ppm, the FAA wished to have its standard in harmony with OSHA's standard of 0.1 ppm.

² A TLV is the time-weighted average concentration for a normal 8-hour workday and a 40-hour workweek, to which workers may be exposed, day after day, without adverse health effect.

8.3.2 Health Effects of Ozone

Extensive investigations of ambient air ozone in humans and experimental animals have been described in several definitive scientific reviews (National Research Council 1977; U.S. Environmental Protection Agency 1986; Lippmann 1989). The health effects are briefly summarized below.

Ozone in the ambient air, in sufficiently high concentrations, irritates the upper respiratory tract, causes measurable degradation of pulmonary function, enhances lung infectivity, and causes alterations in blood biochemistry related to immune response. Most of the reported effects were observed after administration of doses considerably higher than those to which humans are routinely exposed. Under these conditions, morphological effects of ozone on the respiratory tract include damage to ciliated cells, proliferation of bronchiolar cells, cellular inflammation, and thickening of pulmonary arteriolar walls. Short-term exposure to ozone affects pulmonary function by increasing the breathing frequency, various physiological measures of breathing volume, airway resistance, and airway reactivity. Tidal volume, lung compliance, and diffusion capacity are decreased. Long-term exposure to ozone causes increased lung volume and airway resistance, and decreased lung compliance, respiratory flow, and lung function indicators (e.g., FEV₁). Biochemically, ozone causes increases in metabolic enzymes in lung and blood, permeability changes in the lung, and increased oxygen consumption. Finally, ozone affects host defense mechanisms by delaying mucociliary clearance, accelerating alveolar clearance, inhibiting bacterial activity, altering lung macrophages causing a decrease in function, altering the number of defense cells, increasing susceptibility to bacterial infection, and altering immune activity.

Currently at issue is whether exposure to low levels of ozone manifests any of these effects. Recent work was conducted by Horstman et al. (1989), who exposed humans to 0.08 ppm in six 50-minute cycles during exercise representative of a day of moderate or heavy work. At this

level, often found in ambient air, clinically meaningful alterations in lung function were observed. The Clean Air Scientific Advisory Board of the EPA is divided on the implications of these findings for regulation. Nevertheless, this and other recent research is leading to a reexamination of the bases for current regulatory standards and the durations of exposure prescribed in those standards. One of the more prominent issues is the need for an 8-hour ambient air standard for ozone. Such reconsiderations are applicable to the airliner cabin environment, particularly for cabin crew members engaged in the equivalent of moderate exercise at altitude for extended periods of time.

8.3.3 Comparison of Ozone Levels Measured in Airliner Cabins with Existing Standards

A summary of ozone levels measured on all flights in this investigation was presented in Table 4-28. Average concentrations, obtained by integrated sampling, were 0.010 ppm on smoking flights and 0.022 ppm on nonsmoking flights; the maximum concentrations measured among all flights sampled was 0.078 ppm. Concentrations appeared to be uniformly distributed throughout the cabin, precluding the need to consider weighted exposures of cabin crew members and passengers by cabin section. All values were consistently below flight, occupational, and environmental standards established by the Federal government, as indicated in Section 8.3.2. This and current scientific knowledge lead to the conclusion that ozone does not pose a health hazard to cabin crew members or passengers.

8.4 REFERENCES

Benenson, A.S. 1985. Control of Communicable Diseases in Man. Fourteenth Edition. American Public Health Association. Washington, D.C.

Bitton, G. 1980. Introduction to Environmental Virology. John Wiley & Sons, Inc. New York, N.Y.

Bloch, A.B., W.A. Orenstein, W.M. Ewing, W.H. Spain, G.F. Mallison, K.L. Herrman and A.R. Hirman. 1985. "Measles outbreak in a pediatric practice: airborne transmission in an office setting." Pediatrics 75:676-683.

Brockett, R.M. and J.K. Ferguson. 1975. "Microbiological sampling of the spacecraft atmosphere during a simulated Skylab mission." Aviat. Space Environ. Med. 46:30-32.

2028397417

Brackett, R.M., J.K. Ferguson and M.R. Henney. 1978. "Prevalence of fungi during Skylab missions." Appl. Environ. Microbiol. 36:243-246.

Burge, H.A. 1985. "Indoor sources for airborne microbes in indoor air and human health." In: Indoor Air and Human Health. Gammage, R.B. and S.V. Kaye, eds. Lewis Publishers, Inc. Chelsea, MI.

Burge, H.A., M. Chatigny, J. Feeley, K. Kreiss, P. Morey, J. Otten and K. Peterson. 1987. "Guidelines for assessment and sampling of saprophytic bioaerosols in the indoor environment." Appl. Indust. Hyg. 2(5):R10.

Dixon, W.J. and F.J. Massey, Jr. 1969. Introduction to Statistical Analysis, 3rd edition. McGraw-Hill Book Company. NY.

Federal Aviation Administration. 1979. Effects of Ozone on Exercising and Sedentary Adult Men and Women Representative of the Flight Attendant Population. Report of the Civil Aeromedical Institute, Oklahoma City. October 1979. National Technical Information Service, Springfield, Va. FAA-AM-79-20.

Federal Aviation Administration. 1980. Effects of Ozone (0.30 part per million, ~600 ug/m³) on Sedentary Men Representative of Airline Passengers and Cockpit Crewmembers. Report of the Civil Aeromedical Institute, Oklahoma City. March 1980. National Technical Information Service, Springfield, Va. FAA-AM-80-9.

Federal Aviation Administration. 1989. Radiation Exposure of Air Carrier Crewmembers. AAM-624. Federal Aviation Administration, U.S. Department of Transportation.

Federal Register. 1980. Airplane Cabin Ozone Contamination. 45 FR 3880. January 21, 1980.

Feeley, J.C. 1985. "Impact of indoor air pathogens on human health." In: Indoor Air and Human Health. Gammage, R.B. and S.V. Kaye, eds. Lewis Publishers, Inc. Chelsea, Mich.

Horstman, D., W. McDonnell, L. Folinsbee, S. Abdul-Salaam, and P. Ives. 1989. Changes in pulmonary function and airway reactivity due to prolonged exposure to typical ambient ozone (O₃) levels. In: Atmospheric Ozone Research and its Policy Implications. Pages 755-762. T. Schneider et al. (eds.). Elsevier Science Publishers B.V., Amsterdam.

Joklik, W.K. 1985. Virology. Second Edition. Appleton-Century-Crofts. Norwalk, Conn.

Kozak, P.P., J. Gallup, L.H. Cummins and S.A. Gillman. 1979. "Factors of importance in determining prevalence of indoor molds." Ann. Allergy 43:88-94.

Kozak, P.P. and J. Gallup. 1984. "Endogenous mold exposure: environmental risk to atopic and nonatopic patients." In: Indoor Air and Human Health. Gammage, R.B. and S.V. Kaye, eds. Lewis Publishers, Inc. Chelsea, Mich.

Kreiss, K. and M.J. Hodgson. 1984. "Building-associated epidemics." In: Indoor Air Quality. Pages 87-106. Walsh, P.J., C.S. Oudney and E.D. Copenhaver, eds. CRC Press. Boca Raton, Fla.

Letts, R.M. and E. Doermer. 1983. "Conversation in the operating theater as a cause of airborne bacterial contamination." J. Bone Joint Surg. 65:357-362.

Lippmann, M. 1989. Health effects of ozone. A critical review." J. Air Pollut. Control Assoc. 39:672-692.

Loosli, C.G., H.M. Lemon, O.H. Robertson and E. Appel. 1943. "Experimental airborne influenza infection: I. Influence of humidity on survival of virus in air." Proc. Soc. Exp. Biol. Med. 53:205-206.22

Morey, P.R. and J.C. Feeley. 1988. "Microbiological aerosols indoors." ASTM Standardization News (December).

National Research Council. 1986. The Airliner Cabin Air Environment. Air Quality and Safety. National Academy Press. Washington, D.C.

National Research Council. 1977. Ozone and Other Photochemical Oxidants. National Academy Press. Washington, D.C.

Platts-Mills, T.A., F. Rawle and M.D. Chapman. "Problems in allergen standardization." Clin. Rev. Allergy 3:271-290.

Richardson, J.H. and W.E. Barkley. 1984. Biosafety in Microbiological Laboratories. Centers for Disease Control and National Institutes of Health. U.S. Department of Health and Human Services. HHS Publication No. (CDC) 84-8395. Washington, D.C.

Robinson, P.A., R.V. Tauxe, W.G. Winkler and M.E. Levy. 1983. "Respiratory illness in conference participants following exposure to rug shampoo." Infection Control. 4:158-160.

Solomon, W.R. 1976. "A volumetric study of winter fungus prevalence in the air of midwestern homes." J. Allergy Clin. Immunol. 57:46-55.

Spendlove, J.C. and K.F. Fannin. 1983. "Source, significance and control of indoor microbial aerosols: human health aspects." Public Health Rep. 98:229-244.

Tyndall, R.L., C.S. Dudney, A.R. Hawthorne, R. Jernigan, K. Ironside and P. Metler. 1987. "Microflora of the typical home." In: Proceedings of the 4th International Conference on Indoor Air Quality and Climate, Seifert, B., H. Esdorn, M. Fischer, H. Ruden and J. Wegner, eds. Institute for Water, Soil and Air Hygiene. Berlin (West), August 17-21. Volume 1. Pages 617-621.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 1988. Sources, Effects and Risks of Ionizing Radiation. Report to the General Assembly. Annex A: Exposures from natural sources of radiation. UN Publication Sales No. E.88.IX.7. United Nations. New York, N.Y.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 1986. Genetic and Somatic Effects of Ionizing Radiation. Report to the General Assembly. Annex C: Biological effects of pre-natal irradiation. UN Publication Sales No. E.86.IX.9. United Nations. New York, N.Y.

United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 1982. Ionizing Radiation: Sources and Biological Effects. Report to the General Assembly. Annex B. Paragraph 10. UN Publication Sales No. E.82.IX.8. United Nations. New York, N.Y. As cited in United Nations Scientific Committee on the Effects of Atomic Radiation (UNSCEAR). 1988. Sources, effects and risks of ionizing radiation. Report to the General Assembly.

U.S. Environmental Protection Agency. 1986. Air Quality Criteria for Ozone and Other Photochemical Oxidants. Volumes 4 and 5. EPA/600/8-84/0206F. Environmental Criteria and Assessment Office, Research Triangle Park, N.C.

U.S. National Institute for Occupational Safety and Health. 1987. Guidance for Indoor Air Quality Investigations. Hazards Evaluations and Technical Assistance Branch, Division of Surveillance, Hazard Evaluations and Field Studies. Cincinnati, Oh.

Watkins, H.M.S. 1970. "Epidemiologic investigations in Polaris submarines." In: Aerobiology: Proceedings of the Third International Symposium on Aerobiology, Silver, I.H. (ed.). University of Sussex, England. September 1969. Academic Press. New York, N.Y.

Section 9.0

MITIGATION

2028397421

Section 9.0

MITIGATION

As identified through the risk assessment given in preceding sections, the pollutants that pose the highest risks of mortality and morbidity to airliner flight attendants and passengers are ETS contaminants and cosmic radiation. The measurement results also indicated that carbon dioxide levels on flights monitored during this study were frequently above the level thought to satisfy comfort criteria. A general framework for identifying and assessing alternative mitigation strategies for these pollutants is presented in Section 9.1. Application of this framework to strategies for reducing ETS levels in aircraft is described in Section 9.2, and application of the framework to other pollutants (cosmic radiation and carbon dioxide) is described in Section 9.3.

9.1 GENERAL FRAMEWORK FOR ASSESSING MITIGATION OPTIONS

A general framework for evaluating alternative mitigation strategies for a contaminant in an airliner cabin is depicted in Figure 9-1. The first step in this process is to identify candidate mitigation strategies. Such strategies could include potential technological or procedural solutions to apparent problems; the technological solutions generally involve some type of change in aircraft design or equipment, whereas procedural solutions involve changes in the activities of people aboard the aircraft. Although it may be possible to identify many types of candidate strategies, only a limited number will be feasible from technological or procedural standpoints. As a simple example, addition of lead shielding could be contemplated to reduce cosmic radiation exposure, but such a procedure would be technologically impractical because of the resultant increase in aircraft mass. Some qualitative judgments obviously are required in this feasibility assessment process.

In the second step of the overall framework, strategies that survive the feasibility assessment are subjected to a more quantitative process of modeling and estimation. In performing this evaluation, it must

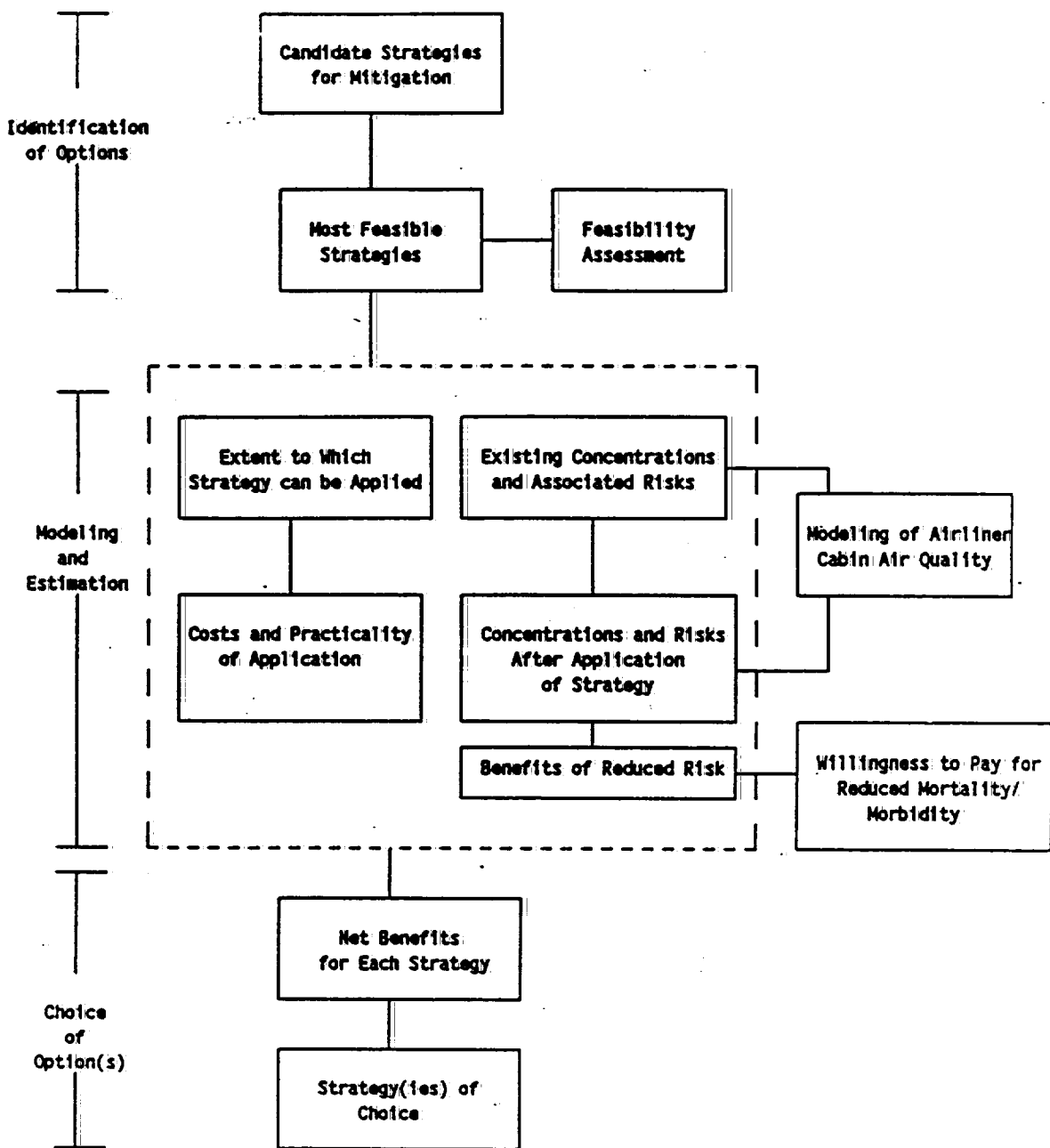


FIGURE 9-1. GENERAL FRAMEWORK FOR ASSESSMENT OF ALTERNATIVE MITIGATION STRATEGIES

be recognized that certain options will have practical upper limits (e.g., extent to which ventilation rates or filter efficiencies can be increased). Some aspects of cost estimation will require detailed pricing or econometric models that cannot be developed within the scope of this effort. In addition to the types of costs (e.g., fuel penalties, new equipment) that can be addressed quantitatively, practical considerations such as anticipated acceptability by airline management, flight crews, or passengers need to be addressed qualitatively. The cost and practical aspects are juxtaposed with the estimated benefits of each strategy. Benefits accrue from presumed decreases in contaminant concentrations and associated health or discomfort risks. The contaminant concentrations expected to prevail when a specific strategy is applied are estimated through cabin air quality modeling, discussed later. The risk reduction associated with reduced concentrations is estimated through the framework used to assess risks for currently prevailing concentrations (see Section 6.0). The benefits of reduced risk can be placed in monetary terms using estimates of an individual's willingness to pay for reduced mortality or morbidity. Such estimates, which are discussed in more detail later, can be taken from other studies.

The third step of the overall framework is to determine the strategy or strategies of choice. Generally speaking, the optimal strategy would be the one with the highest net benefit (i.e., benefit minus cost), given cost and distributional constraints. However, if two candidate strategies have similar estimates for risk reduction, then a cost-effectiveness analysis can be performed, with a focus on the costs and practical aspects of each alternative.

9.2 APPLICATION OF FRAMEWORK TO ETS CONTAMINANTS

The framework described above is first applied to ETS contaminants. Alternative mitigation options that are considered, and the subset retained for further analysis, are discussed in Section 9.2.1. Modeling efforts and estimated costs and benefits for each strategy are described in Section 9.2.2. Discussion of the relative costs and benefits of the alternative strategies is provided in Section 9.2.3.

9.2.1 Identification of Options

Eight candidate options for reducing the exposure of flight attendants or passengers are identified in Table 9-1. Half the options require a technological approach and the other half require a procedural approach. The options are also classified according to three general types of strategies for mitigating potential exposures:

- (1) Preventing or minimizing the emission of ETS contaminants from cigarettes (i.e., emissions reduction)
- (2) Removing the ETS contaminants from the cabin environment after they have been introduced (i.e., contaminant removal)
- (3) Reducing the exposure of cabin occupants to ETS contaminants that have been introduced (i.e., exposure management).

Although some of these options could obviously be used in combination with others, the general feasibility of each option has been assessed separately, as discussed below.

For the strategy involving emissions reduction, an obvious option is an outright ban of smoking on all flights. This procedural option would be quite feasible to implement and, in fact, has been implemented in partial form on domestic flights (i.e., smoking not allowed for flights of two hours duration or less under Public Law 100-202)¹. Consideration would need to be given to possibilities such as smokers experiencing withdrawal symptoms, becoming unruly, or attempting to smoke in the lavatory, thereby creating additional hazards for other passengers.

A different type of procedural option would involve curtailment of smoking by restricting the periods when it is allowed. For example, smoking could be allowed for a period of 10 minutes after every two hours of flight time, consistent with the earlier ban for flights shorter than

¹All of the work described in the report preceded passage of PL101-164, which will ban smoking on all domestic commercial flights under six hours in duration.

TABLE 9-1. MAJOR OPTIONS CONSIDERED FOR MITIGATION
OF ETS CONTAMINANTS

General Strategy	Approach Required	
	Technological	Procedural
1. Reduction of emissions		
- ban on smoking (total or partial)		X
- curtailment of smoking period		X
2. Contaminant removal/confinement		
- increased ventilation	X	
- local exhaust (smoking section)	X	
- smoking lounge	X	
- filtration/sorption	X	
3. Exposure management		
- separate smoking/nonsmoking flights		X
- stationing of flight attendants		X

two hours. In this case, consideration would need to be given to the possibility of substantially elevated ETS levels during the smoking period, since most smokers would probably smoke during this time.

The next set of options to be evaluated involves the notion of removal of ETS contaminants, rather than reduction of emissions. The rate of removal could be increased, for example, by increasing the amount of fresh-air intake to the airliner cabin. The extent to which fresh air could be added has a practical upper limit, however, related to the need to maintain a prescribed cabin pressure. An added benefit of this approach would be reduction of levels of some other pollutants (e.g., carbon dioxide) having sources within the cabin environment. Potential disadvantages could include the added fuel penalty associated with increased fresh-air intake, the need for increased thermal treatment of incoming air, potential increases in ozone levels, and potential decreases in relative humidity levels. The extent of contaminant removal due to increased ventilation can be modeled, and the associated fuel penalty can be estimated. Because both the strategy and the modeling of consequences are feasible, this option can be subjected to a quantitative assessment.

Local exhaust can be thought of as a special case of increased ventilation. This strategy would be most effective if combined with the concept of a smoking lounge, discussed below. Under the current configuration of smoking and no-smoking sections, the notion of local exhaust would essentially be tantamount to increasing the fresh-air supply to the smoking section only. Although there are some uncertainties related to technological feasibility and costs, the strategy is feasible and its potential consequences can be modeled.

Another special case of increased ventilation would involve the creation of a smoking lounge, which would also serve to confine ETS emissions. In a simplified form, this option would involve creating smoking sections of fixed size with physical barriers (e.g., walls/door or curtains) separating such compartments from nonsmoking sections. This option, while technologically feasible, would be inefficient (1) because

of the need to create smoking/nonsmoking sections of fixed size, as opposed to the concept of a "sliding" boundary that is currently used to accommodate varying numbers of nonsmokers on smoking flights, and (2) because it would do little to reduce the risks of flight attendants assigned to or passing through the smoking section (as shown in Section 7.0, risks related to ETS exposures are estimated to be highest for flight attendants).

A truer version of the smoking-lounge concept would be construction of an actual lounge on one side of the plane toward the rear. This lounge could be "visited" by smokers wishing to smoke, much in the same sense as lavatories are currently visited by cabin occupants. The size of the lounge (and maximum occupancy) would obviously need to be limited, and emissions could be effectively confined by providing an independent exhaust system for the lounge. Flight attendants would not need to enter the lounge, thereby minimizing their exposures. Some challenges in design and financing of the lounge, however, would be likely. Some of the costs could be recovered by charging a per-visit fee for the lounge. However, this approach would add some administrative burden, and the extent of costs recovered (both the cost of building the lounge and the cost of reduced seating capacity) would be somewhat difficult to predict. Thus, while potentially attractive, concepts for emissions confinement should be dismissed at this time as impractical to implement.

A third type of contaminant-removal option involves improved filtration of particle-phase ETS constituents or sorption of gas-phase constituents. Such an option obviously would be viable only for aircraft with recirculation capabilities, but the percent of aircraft with recirculation is expected to steadily increase in the future. Most aircraft with recirculation are currently equipped with some type of filter in the recirculation loop, and the efficiency of these filters can presumably be improved. Some potential drawbacks of filtration are (1) that filtration of only particle-phase constituents would not remove the gas-phase constituents that can cause odor and irritation, and (2) some gas-phase

constituents, following removal by sorption, could conceivably volatilize and subsequently cause odor/irritation problems throughout the aircraft. Although some uncertainties are involved, modeling can be performed with assumptions involving efficiencies of currently installed filters and the extent of improvement that may be technologically feasible. Like the option of increased fresh-air intake, filtration may also achieve some reduction of pollutants other than ETS contaminants.

The last set of options involves the notion of exposure management rather than emissions reduction or contaminant removal. An extreme example would be to have separate smoking and no-smoking flights. Although such an approach would reduce exposures for nonsmoking passengers, it would not necessarily reduce flight attendants' overall exposures. The model required to assess the economic consequences of separate smoking and nonsmoking flights would be difficult to construct and would involve a number of assumptions. Even without such a model, it seems unlikely that such an approach would be economically viable. Thus, it should be dismissed at this time as impractical and having questionable benefits that cannot easily be modeled.

Another approach to exposure management would involve rotating flight attendants so that each is assigned to the smoking section only for some fraction of flights. This approach, however, would merely redistribute risk; the aggregate risk for flight attendants would not be reduced, and risks for nonsmoking passengers would be unaffected. Thus, the strategy would have no apparent benefits. A variation of this theme would involve recognition rather than reduction of risk. For example, flight attendants stationed in the smoking section could be offered "hazardous duty pay." The costs of increased risk could be estimated, translated into salary differentials, and the costs recovered through differential pricing for smoking and no-smoking seats. Such an approach, however, could affect passenger behavior (e.g., more smoking passengers opting for no-smoking seats, which would reduce ETS levels and associated risks for attendants), thereby adding a layer of assumptions and uncertainties to

the assessment. Like the other options for exposure management, no benefits would accrue to nonsmoking passengers (unless ETS levels would actually decrease through this approach). Thus, approaches involving exposure management can be dismissed as having very limited benefits and posing some difficulties in econometric modeling needed to help determine the extent of any potential benefits.

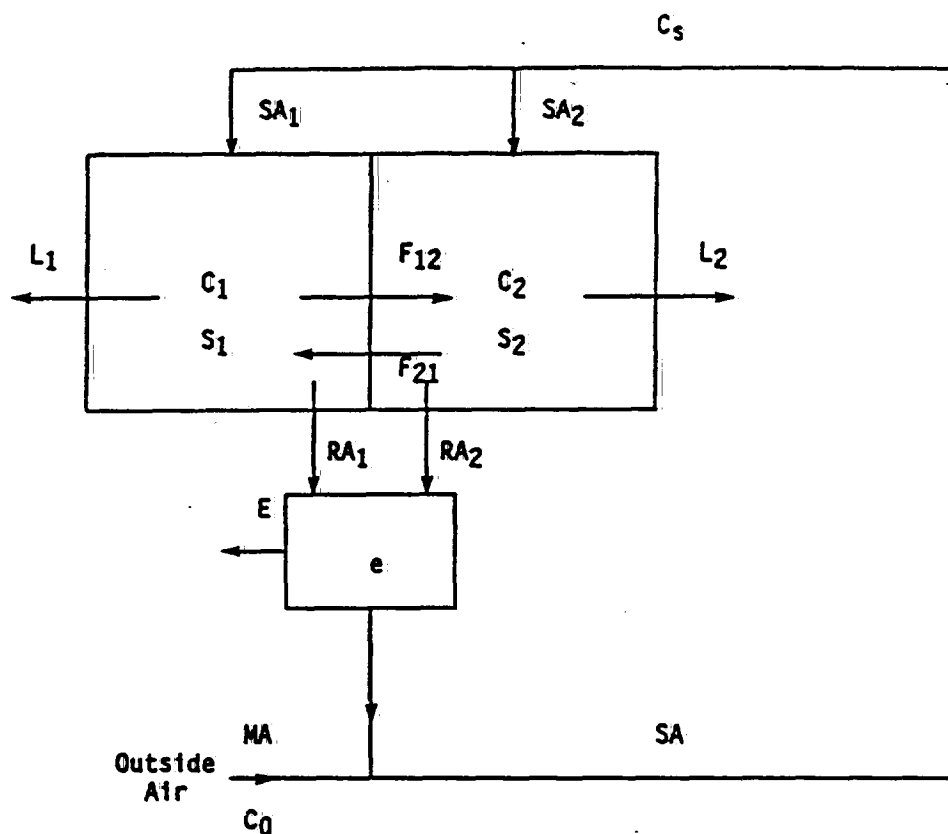
Based on the above discussion, the following candidate approaches to ETS mitigation have been retained for further, quantitative analysis:

- Ban on smoking (total or partial)
- Curtailment of smoking period
- Increased intake of fresh air (including special case targeted at smoking section)
- Filtration/sorption of ETS contaminants.

9.2.2 Modeling of Cabin Air Quality

Model Description. Air quality modeling was performed to assess the potential impacts of alternative mitigation strategies on ETS concentrations in cabin environments. The focus of the modeling effort was on RSP, which was used as the ETS tracer in performing the risk assessment for chronic effects due to ETS exposure. A two-chamber model, depicted in Figure 9-2, was developed; this model, similar in concept to that described by Ryan et. al (1988), treats the smoking and no-smoking sections as separate compartments with communicating airflows. The model also allows contaminant emission rates to be specified for each compartment and incorporates supply airflow rates from fresh (makeup) air and recirculated air (where applicable) as well as return airflow from each compartment that is exhausted from the aircraft or recirculated.

The model can actually be thought of as a three-chamber model, with the supply airstream representing the third chamber. Under steady-state conditions (appropriate for predicting average concentrations



Legend

- C indicates contaminant concentrations (mg/m^3)
- S indicates emission rate (mg/h)
- F indicates interchamber flow rate (m^3/h)
- L indicates leakage rate (m^3/h)
- RA indicates return-air flow rate (m^3/h)
- e indicates filter efficiency (dimensionless fraction, $0 \leq e \leq 1$)
- E indicates exhaust flow rate (m^3/h)
- MA indicates makeup-air flow rate (m^3/h)
- SA indicates supply-air flow rate (m^3/h)
- Subscripts 0, s, 1 and 2 refer to outdoors, supply airstream, chamber 1, and chamber 2

FIGURE 9-2. SCHEMATIC OF TWO-CHAMBER MODEL FOR CABIN AIR QUALITY

related to chronic health effects), the contaminant mass balance for each chamber is as follows (the terms used below are defined in Figure 9-2):

$$C_S \cdot SA = C_0 \cdot MA + (1 - e) \cdot (C_1 RA_1 + C_2 RA_2) / (RA_1 + RA_2) \cdot (RA_1 + RA_2 - E)$$

$$C_S \cdot SA_1 + C_2 \cdot F_{21} + S_1 = C_1 (L_1 + RA_1 + F_{12})$$

$$C_S \cdot SA_2 + C_1 \cdot F_{12} + S_2 = C_2 (L_2 + RA_2 + F_{21})$$

The above mass-balance description yields a system of three equations and three unknowns (C_S , C_1 , and C_2) which can be obtained by solving the equations simultaneously. In solving the equations, fresh-air supply rates and interchamber airflow rates were based on PFT measurements. Leakage rates were assumed to equal zero because no quantitative guidance was available for specifying these rates; thus, any leakage is captured in the term for exhaust flow rate, equal to the fresh-air intake rate by assumption. The return airflow rate incorporates recirculation airflow rates based on aircraft specifications (Lorengo and Porter, 1985). A filter efficiency of 90 percent for RSP removal was assumed for baseline modeling, based on information reported by Lorengo and Porter (1985). An emission factor of 26 mg/cigarette (NRC, 1986) was combined with technician observations of smoking rates to develop an hourly emission rate for each flight that was modeled. Supply airflow rates for each section of the aircraft were apportioned by volume, using the number of rows in each section as a proxy for volume. Return airflow rates were determined by flow-balance considerations, given supply and interchamber airflow rates.

Although PFTs were deployed to estimate airflow rates on study flights, practical limitations (i.e., the need for unobtrusive measurements) precluded obtaining meaningful measurement results in a number of cases. Ideally, PFT sources and samplers would have been distributed throughout each section (smoking and no-smoking) of the aircraft; however, logistical constraints restricted the approach to one release location and one sampling location per section. PFT measurement results were reviewed

to determine cases for which results were most plausible, according to the following criteria:

- (1) Measured ventilation rates for the aircraft determined by two different PFT methods (single tracer common to both sections and tracers unique to each section) were consistent with one another and with maximum ventilation rates indicated by aircraft specifications.
- (2) Interzonal airflow rates were positive but not excessively large.

The three flights chosen for modeling involved two types of narrow body aircraft (B-727 with no recirculation and MD-80 aircraft with recirculation) that collectively accounted for more than 50 percent of the flights monitored during the study. Selected characteristics of the aircraft and flights used for RSP modeling are given in Table 9-2. The flights collectively provide a ten-to-twenty-fold range in smoking rates and measured ETS concentrations in the smoking section.

The model described previously was chosen over the one developed by Ryan et al. (1988) because of the ability to include a filtration factor for recirculated air (important to the analysis of mitigation options related to filtration/sorption). However, the software for the Ryan et al. (1988) model was obtained from the principal author and applied to the case without recirculation that was listed in Table 9-2. The published model and the model developed specifically for the mitigation assessment yielded identical results when applied to this case.

Model Application. Results of baseline modeling for the three study flights, to be used as a benchmark for assessing various mitigation alternatives, are compared with measured RSP concentrations in Table 9-3. Although the modeling results are generally lower than measured values, the general patterns of results are consistent. For example, both measured and modeled values indicate somewhat greater migration of RSP from the smoking to the no-smoking section for the MD-80 than for the B-727 aircraft, presumably due to air recirculation. Both the

TABLE 9-2. SELECTED CHARACTERISTICS OF FLIGHTS/AIRCRAFT
USED FOR RSP MODELING

Characteristic/Model Input	Flight		
	1	2	3
Type of Aircraft	B-727	MD-80	MD-80
Number of Passenger Rows (Number assigned to coach smoking)	21 (2)	33 (7)	38 (8)
Passenger Capacity	108	142	142
Observed Smoking Rate (cigarettes/h)	3	1	15
Measured Fresh-air Intake Rate, m ³ /h	3,579	3,125	3,964
Percent Recirculation Air*	0	21	21
Chamber Airflow Rates, m ³ /h			
- SA ₁	3238.0	3116.9	3960.9
- SA ₂	340.8	839.2	1056.2
- RA ₁	3058.5	3636.5	4036.6
- RA ₂	520.3	319.6	980.5
- F ₁₂	308.1	434.0	859.8
- F ₂₁	128.6	953.6	935.5

* Per aircraft specifications

TABLE 9-3. MEASURED AND MODELED* RSP CONCENTRATIONS FOR
THREE STUDY FLIGHTS

Flight/Section	RSP Concentrations, $\mu\text{g}/\text{m}^3$	
	Measured	Modeled
Flight 1 (B-727)		
- no-smoking section**	31.9	4.7
- smoking section	233.5	122.4
Flight 2 (MD-80)		
- no-smoking section**	11.0	5.3
- smoking section	7.3	22.3
Flight 3 (MD-80)		
- no-smoking section**	86.3	44.2
- smoking section	302.0	224.3

* Baseline model, derived from measurements together with assumed recirculation rate of 21 percent and filter efficiency of 90 percent for MD-80 aircraft.

** Volume-weighted average of gravimetric and optical measurements in boundary, middle, and remote locations.

measured and modeled values also have some uncertainties; in the case of modeled values, sources of uncertainty include emission, mixing, and deposition rates, fresh-air supply and interchamber airflow rates, the prevailing recirculation rate during a flight, and the filter efficiency for RSP removal.

As noted in Section 9.2.1, four alternatives for ETS mitigation were retained for further analysis:

- Ban on smoking (total or partial)
- Curtailment of the smoking period
- Increased ventilation (including special case targeted at smoking section)
- Filtration/sorption of ETS contaminants.

The total ban on smoking requires no modeling; if this option were exercised, then RSP levels on current smoking flights would be reduced to those prevailing on non-smoking flights, and the incremental exposure and incremental risk would be zero. Similarly, modeling is not required to assess the impact of partial bans; population exposures to ETS-related RSP would be reduced essentially in proportion to the reduction in number of flight hours during which smoking would be permitted (the reduction would not be exactly proportional because longer flights generally have larger aircraft capacities, greater percentages of time when the no-smoking light is not illuminated, and possibly different smoking rates than shorter flights).

A data file supplied by DOT, containing information on all flights scheduled for departure from U.S. airports during January 1989, was analyzed to determine the relative frequencies for domestic flights of different durations. The analysis was based on jet aircraft flights departing from 70 airports associated with large and medium air traffic hubs, consistent with the sampling frame used for the study (see Section 2.4). The relative frequencies of flights and flight hours represented by

flights of different durations (classified into hourly duration intervals) are summarized in Table 9-4. Flights under two hours in duration account for 44.5 percent of all flight hours. Thus, under the two-hour ban enacted in April 1988 under PL 100-202, smoking would be allowed during 55.5 percent of all flight hours. (A more detailed analysis, factoring in the specific policies of Northwest Airlines and United Airlines, indicated a revised figure of 54.3 percent.) A four-hour ban would limit smoking to 14 percent of all flight hours, and a six-hour ban would restrict smoking to 2 percent of all flight hours, as illustrated in Figure 9-3.

Two hypothetical scenarios were examined for curtailment of the smoking period:

- Restriction of smoking to a 10-minute period after every two hours of flight time
- Restriction of smoking to a 10-minute period after every hour of flight time

The impact of each scenario on the smoking rate (cigarettes per flight) was estimated for each domestic smoking flight monitored during the study by assuming that each passenger seated in the smoking section would smoke one cigarette during each period when smoking was allowed. On the average, the first scenario would lower total smoking per flight by about 70 percent (i.e., from 51.9 to 15.2 cigarettes/flight) and the second scenario would reduce total smoking by about 25 percent (from 51.9 to 39.8 cigarettes/flight). Each of the flights previously chosen for modeling was modeled with these reductions in the smoking rate. As shown in Table 9-5, the reduction in average RSP concentrations in both the no-smoking and the smoking sections was proportional to the reduction in smoking rate in all three cases. However, as noted earlier, short-term peaks in RSP and gas-phase ETS constituents could rise sharply if smoking periods were restricted, thereby increasing irritation and discomfort for flight attendants and passengers.

The impact of the increased fresh-air intake rates was first examined for the flight with the highest smoking rate (flight 3).

TABLE 9-4. RELATIVE FREQUENCIES FOR DOMESTIC FLIGHTS
OF DIFFERENT DURATIONS

Flight Duration	Percentage of Flights	Percentage of Flight Hours
< 1 hour	17.6	7.4
1-1.99 hours	48.7	37.1
2-2.99 hours	21.3	28.1
3-3.99 hours	7.2	13.4
4-4.99 hours	3.2	7.6
5-5.99 hours	1.5	4.3
≥ 6 hours	0.6	2.1
Total, all durations	100.0	100.0

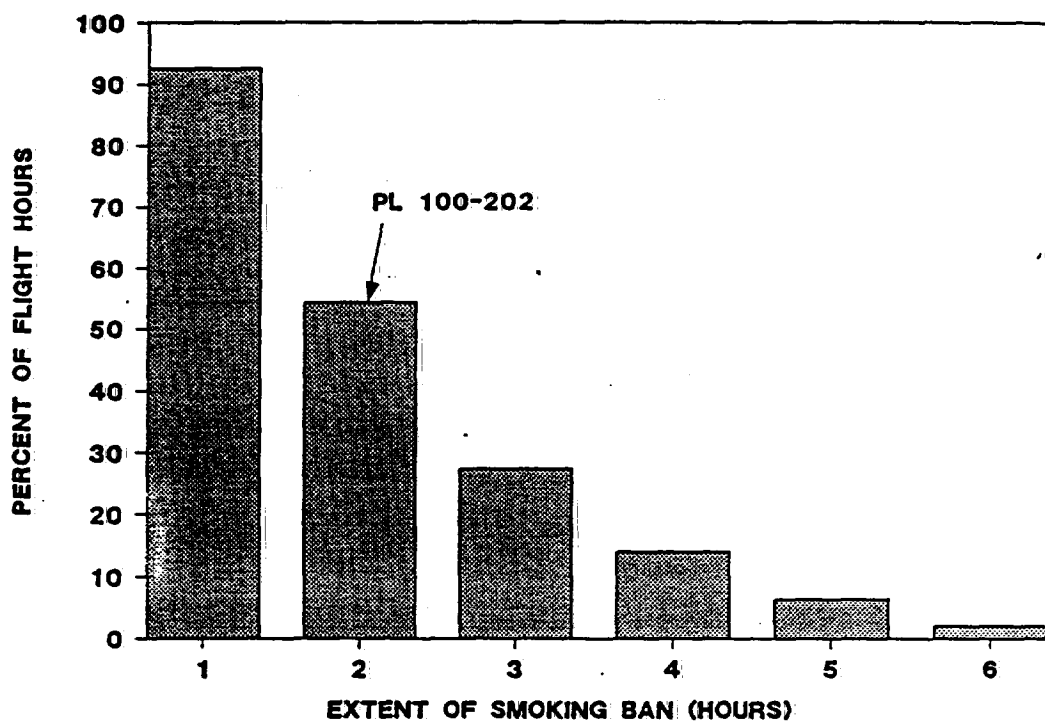


FIGURE 9-3. PERCENT OF FLIGHT HOURS DURING WHICH SMOKING WOULD BE PERMITTED UNDER PARTIAL SMOKING BANS RELATED TO FLIGHT DURATION

TABLE 9-5. PREDICTED RSP CONCENTRATIONS FOR THREE STUDY FLIGHTS
WITH HYPOTHETICAL REDUCTIONS IN SMOKING DUE TO
CURTAILMENT OF SMOKING PERIODS

Case Modeled	RSP Concentration, $\mu\text{g}/\text{m}^3$	
	No-smoking Section	Smoking Section
<u>Flight 1 (B-727)</u>		
- no curtailment (base case)	4.7	122.4
- ten-minute smoking period every hour (total smoking reduced by 25 percent)	3.5 (25%)*	91.8 (25%)
- ten-minute smoking period every two hours (total smoking reduced by 70 percent)	1.4 (70%)	36.7 (70%)
<u>Flight 2</u>		
- no curtailment (base case)	5.3	22.3
- ten-minute smoking period every hour (total smoking reduced by 25 percent)	4.0 (25%)	16.8 (25%)
- ten-minute smoking period every two hours (total smoking reduced by 70 percent)	1.6 (70%)	6.7 (70%)
<u>Flight 3</u>		
- no curtailment (base case)	44.2	224.3
- ten-minute smoking period every hour (total smoking reduced by 25 percent)	33.2 (25%)	168.2 (25%)
- ten-minute smoking period every two hours (total smoking reduced by 70 percent)	13.3 (70%)	67.3 (70%)

* Numbers in parentheses indicate percent reduction in concentration from the base case.

Hypothetical increases of 25, 50, 75 and 100 percent in fresh-air intake were modeled. The results displayed in Figure 9-4 indicate a curvilinear relationship between increase in fresh-air intake and RSP concentration in either section; however, the relationship is more direct than indicated--when the intake rate is doubled, the concentrations are halved. Thus, for example, to reduce concentrations by an order of magnitude, a tenfold increase in fresh-air intake would be required. However, such an increase is not likely achievable, and resultant airflows in the cabin would cause intolerable drafts for passengers. In addition, as noted earlier, ozone concentrations in the cabin could increase and relative humidity levels could decrease.

The impact of a more likely achievable 50-percent increase in the fresh-air intake rate is shown for each of the three modeled flights in Table 9-6. In each case, concentrations in both the no-smoking and smoking sections are reduced by one-third; that is, the concentrations with a 50-percent increase in fresh-air intake are two-thirds of their original values, consistent with the ratio of the old-to-new intake rate (i.e., $1/1.5$ or 0.67).

A special case of increased fresh air is increasing the amount supplied to the smoking section only. If the fresh air supplied to the smoking section is increased by 50 percent, the overall increase is only 10.5 percent (because the smoking section accounts for only 21 percent of the total airflow). Although the reduction in RSP concentrations (23 percent, as shown in the bottom portion of Table 9-6) is less than that achieved with a 50-percent increase in fresh air to the entire cabin, the relative effectiveness is greater; the ratio of concentrations is 0.77 (i.e., $34.2/44.2$ for the no-smoking section and $172.6/224.3$ for the smoking section) whereas the ratio of infiltration rates for the aircraft is 0.9 ($1/1.105$). An assumption in modeling this case was that increased air supply to the smoking section would be compensated by increased exhaust from that section; otherwise, the smoking section would be over-supplied, increasing the flow rate from the smoking to the no-smoking

TABLE 9-6. PREDICTED RSP CONCENTRATIONS FOR THREE STUDY FLIGHTS
WITH A HYPOTHETICAL INCREASE IN THE FRESH-AIR INTAKE RATE

Case Modeled	RSP Concentration, $\mu\text{g}/\text{m}^3$	
	No-Smoking Section	Smoking Section
<u>Flight 1</u>		
- no increase (base case)	4.7	122.4
- 50-percent increase	3.1 (33%)*	81.6 (33%)*
<u>Flight 2</u>		
- no increase (base case)	5.3	22.3
- 50-percent increase	3.6 (33%)	14.8 (33%)
<u>Flight 3</u>		
- no increase (base case)	44.2	224.3
- 50-percent increase	29.5 (33%)	149.5 (33%)
- 50-percent increase for smoking section only	34.2 (23%)	172.6 (23%)

* Numbers in parentheses indicate percent reduction in concentration from the base case.

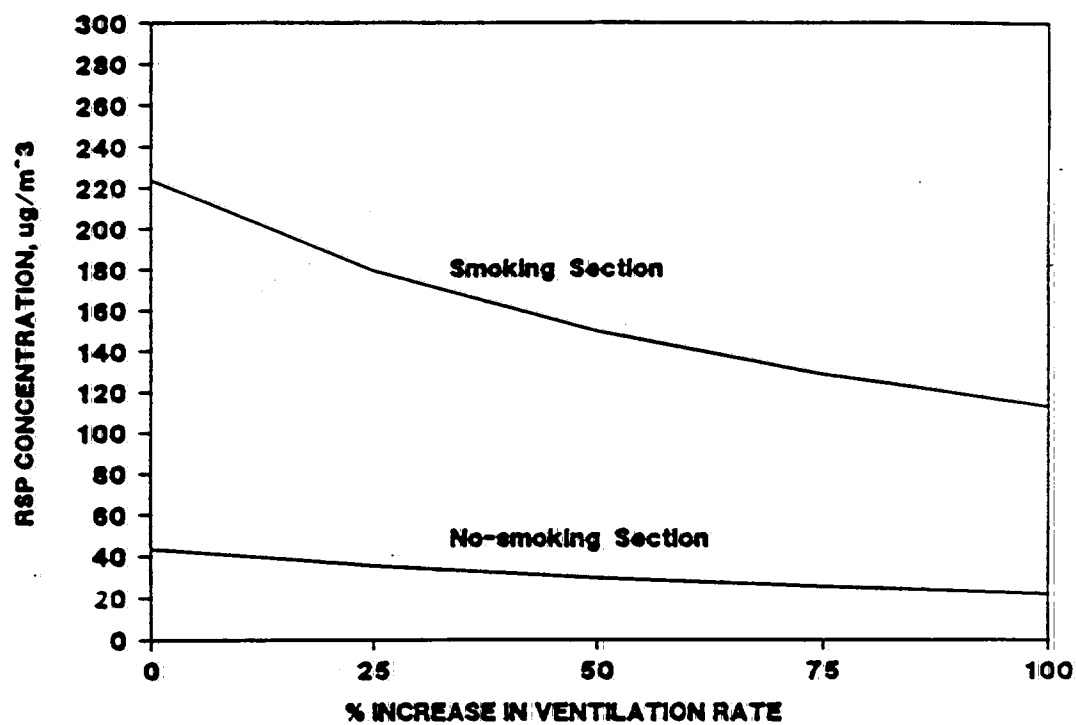


FIGURE 9-4. RELATIONSHIP BETWEEN RSP CONCENTRATIONS AND RATES OF INCREASE IN FRESH-AIR INTAKE FOR ONE STUDY FLIGHT

section. As a result, RSP levels in the smoking section would decrease even further, but levels in the no-smoking section would increase.

The impact of filtration was examined in greatest detail for the flight with the highest smoking rate. The MD-80 aircraft for this flight has a specified air circulation rate of 21 percent (i.e., 21 percent of the air supplied to the cabin is recirculated air). RSP concentrations were modeled with hypothetical filter efficiencies of 0 (i.e., no filter), 0.3, 0.6, 0.8, 0.9, 0.95 and 0.99 (filters currently in use on aircraft are thought to have RSP removal efficiencies in the neighborhood of 0.9). As illustrated in Figure 9-5, overall RSP reductions are less than proportional to filter efficiency, because filtration competes with fresh air for RSP removal and only a fraction of the cabin air is recirculated through the filter. A change in filter efficiency from 0 to 0.99 would reduce RSP concentrations for this flight by 33 percent in the no-smoking section (from 63.2 to 42.7 $\mu\text{g}/\text{m}^3$) and by 8 to 9 percent in the smoking section (from 243.3 to 222.8 $\mu\text{g}/\text{m}^3$).

Increased filter efficiency would provide no benefit for aircraft with no recirculation capability, such as the B-727 for flight 1. For flights 2 and 3 (MD-80 aircraft), the effect of increasing filter efficiency from 90 to 99 percent was modeled. As shown in Table 9-7, minor reductions in RSP (less than 5 percent) would be achieved with more efficient filters. Because some aircraft have higher recirculation rates (up to 50 percent), flight 3 was also modeled with an MD-80 aircraft having a hypothetical recirculation rate of 50 percent. As shown in the lower portion of Table 9-7, RSP concentrations for the base case (90-percent filter efficiency) were slightly higher with 50-percent recirculation than with 21-percent recirculation. The RSP reductions due to improved filter efficiency are projected to be somewhat greater if the aircraft had 50-percent recirculation, but the reductions are still less than 10 percent. Similarly, the RSP reductions due to improved filtration will be somewhat greater if the current filter efficiency is below 90 percent; however, as shown previously in Figure 9-5, the percent reduction due to filtration is relatively insensitive to filter efficiency.

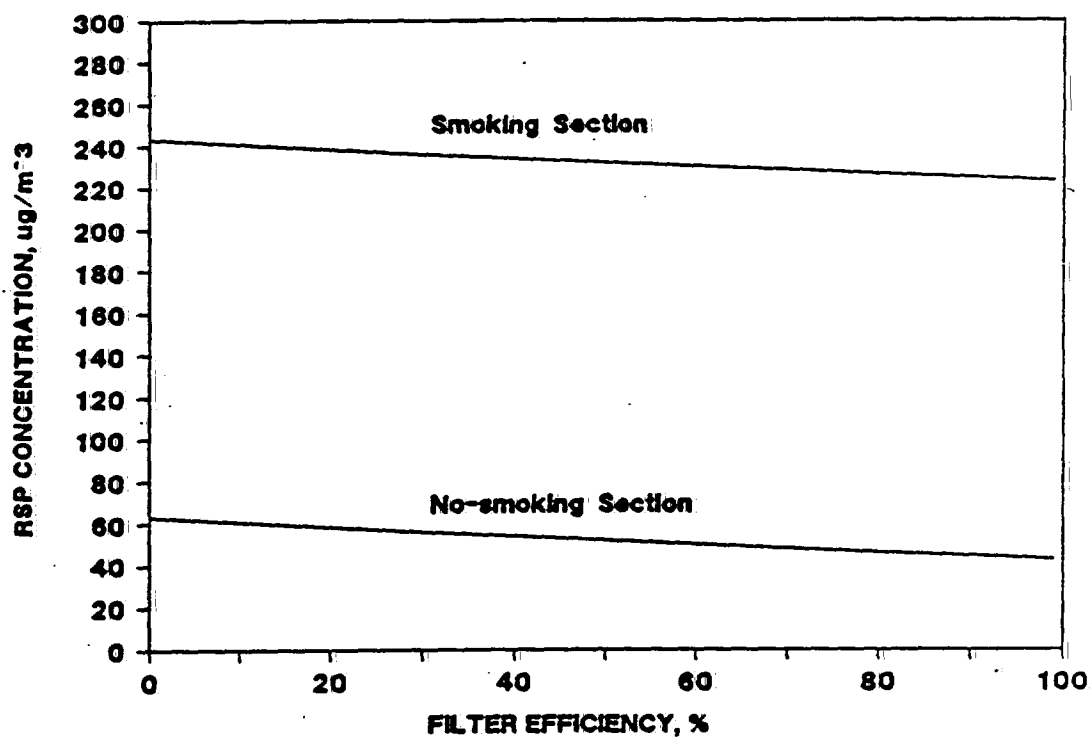


FIGURE 9-5. RELATIONSHIP BETWEEN RSP CONCENTRATIONS AND FILTER EFFICIENCY FOR ONE STUDY FLIGHT WITH 21-PERCENT AIR RECIRCULATION

TABLE 9-7. PREDICTED RSP CONCENTRATIONS FOR TWO STUDY FLIGHTS WITH A HYPOTHETICAL INCREASE IN FILTER EFFICIENCY

Case Modeled	RSP Concentration, $\mu\text{g}/\text{m}^3$	
	No-Smoking Section	Smoking Section
<u>Flight 2 (MD-80 with 21 percent recirculation)</u>		
- 90-percent filter efficiency (base case)	5.3	22.3
- 99-percent filter efficiency	5.2 (2%)*	22.2 (<1%)
<u>Flight 3 (MD-80 with 21 percent recirculation)</u>		
- 90-percent filter efficiency (base case)	44.2	224.3
- 99-percent filter efficiency	42.7 (3%)	222.8 (1%)
<u>Flight 3 (MD-80 with hypothetical recirculation of 50 percent)</u>		
- 90-percent filter efficiency	46.6	226.7
- 99-percent filter efficiency	42.9 (8%)	223.0 (2%)

* Numbers in parentheses indicate percent reduction in concentration from the base case.

9.2.3 Cost-Benefit Analysis

A complete cost-benefit analysis of alternative mitigation strategies would include a full accounting of all categories of costs and benefits. Mitigation costs include not only the cost of the technical approach, but the losses (if any) to smokers required to modify their behavior, and, if appropriate, losses in profits to airlines to the extent that smokers fly less often. Economists would measure losses to smokers as their willingness to pay (WTP) to avoid having their behavior modified. This type of measure has been applied with reasonably high replicability of results in other contexts, but not to the issue of valuing smokers' WTP.

In the analysis below, only technical costs are considered, because of lack of information on the other cost categories. This limitation means that procedural approaches are given zero cost, clearly an underestimate.

On the benefit side, mortality, morbidity, and comfort considerations dominate. Mortality reductions and their associated economic benefits (measured in terms of the WTP for a reduction in the risk of death divided by the given risk reduction) are estimated below. The linkages between passive ETS exposure and morbidity (acute effects, such as eye irritation, exacerbation of chronic conditions, say by helping initiate an asthma attack, and increase in the probability of developing chronic conditions) are not well enough understood to include these effects (although estimates of the WTP for these effects exist in the economics literature). Comfort effects related to odor or other effects that might be part of the WTP of nonsmokers to have their ETS exposure reduced also cannot be included because of data limitations. Because a ban on smoking has to have the largest quantifiable benefit but a zero (quantifiable) cost, it must appear as the best approach, subject to the incomplete analysis.

Benefit calculations for the mitigation analysis focused on reductions in risk of lung cancer mortality due to ETS exposure, using RSP as a tracer. To treat mortality risks in monetary terms, estimates are

needed either for the willingness to pay to avoid specific risks of death or an assumed value of a statistical life (VSL).² The most recent valuation and wage-risk studies provide VSL estimates in the range of \$2 to \$5 million (Viscusi, 1986). A value of \$3.75 million was chosen for this analysis, consistent with recent EPA assessments (Fisher et al. 1987).

Use of the VSL approach requires that the results of the risk assessment be translated into annual expected premature lung cancer deaths due to ETS exposure for the flying population, including both passengers and flight attendants. Based on the estimated cancer risks per 100,000 cabin occupants provided in Section 7.0, estimated annual deaths to be expected in the absence of any ban on smoking for domestic flights are 0.44 for passengers and 0.34 for flight attendants (see Table 9-8). The estimated annual deaths given here are higher than those given in Section 7.0 because the estimates in Table 9-8 assume that smoking would be allowed on all domestic flights, whereas the estimates in Section 7.0 assume that smoking would be allowed only on flights of two-hour or longer durations. Given a VSL of \$3.75 million, the expected deaths in Table 9-8 translate into annual economic values of \$1.65 million and \$1.28 million, respectively. There are also increments in morbidity due to ETS exposure that have not been taken into account.

Projected annual benefits and costs of alternative mitigation options are given in Table 9-9. The greatest benefit (\$2.93 million) would result from a total ban on smoking; benefits other than reduced mortality risk could accrue, for example, from reduced maintenance (e.g., changing of filters) or cleaning costs in the absence of smoking. There are no direct costs of implementing such a ban, although dollar values could conceivably be attached to smokers' inconvenience and discomfort.

² The VSL can be thought of as the average willingness to pay for a given reduction in mortality risk, divided by the risk reduction. Thus, if 1,000 individuals are willing to pay an average of \$2,000 for a 1/1,000 reduction in mortality risk, then the average VSL is \$2 million.

TABLE 9-8. ESTIMATION OF ANNUAL EXPECTED DEATHS DUE TO
PASSENGER AND FLIGHT ATTENDANT EXPOSURES TO ETS
WITH UNRESTRICTED SMOKING ON DOMESTIC FLIGHTS

Passengers

418 million enplanements per year for domestic flights*
 $\times 1.84$ (hours per flight)**
 769 million passenger-hours per year
 $\times .9375$ (fraction of time smoking allowed)***
 721 million passenger-hours per year with smoking permitted
 $+ 45$ (hours per year per flying passenger used in risk assessment)
 16 million people flying 45 hours per year
 $+ 40$ (average lifetime for flying used in risk assessment)
 0.4 million lifetimes of flying 45 per hours per year
 $\times 1.1$ (deaths per million flying lifetimes)
 0.44 expected deaths per year due to ETS exposure

Flight Attendants

56 thousand flight attendants flying 900 hours/year on domestic flights
 $+ 20$ (average lifetime for flying used in risk assessment)
 2.8 thousand lifetimes of flying 900 hours per year
 $\times 0.12$ (deaths per thousand flying lifetimes)
 0.34 expected deaths per year due to ETS exposure

* Source: NRC (1986)

** Based on analysis of data file provided by FAA

*** Assuming no-smoking light is illuminated 6.25 percent of the time

TABLE 9-9. PROJECTED ANNUAL BENEFITS AND COSTS FOR ALTERNATIVE MITIGATION STRATEGIES TO REDUCE ETS EXPOSURES

Strategy	Exposure Reduction	Annual Benefits (\$ million)		Annual Costs (\$ million)
		Passengers	Attendants	
Total ban on smoking	100%	1.65	1.28	0*
Partial ban on smoking				
- flights under two hours	45%	0.74	0.58	0
- flights under six hours	98%	1.62	1.25	0
Curtailment of smoking				
- 10 minutes every 2 hours	70%	1.16	0.90	0
- 10 minutes every hour	25%	0.41	0.32	0
Increased fresh-air intake				
- 50 percent for entire cabin	33%	0.54	0.42	30.8 to 51.5
- 50 percent for coach smoking	23%	0.38	0.29	6.2 to 10.3
Increased filter efficiency** 4-5%*** (from 90 to 99 percent)		0.08	0.06	****

* Assuming that a value can be placed on smokers' inconvenience and discomfort (e.g., willingness to pay for the right to smoke on aircraft), some costs could be estimated; however, no studies to provide such inputs have been identified. Costs could conceivably be estimated for losses in ridership due to smokers opting for other modes of transportation.

** Assuming that all aircraft have recirculation and filters

*** 4.8 percent for passengers, 4.5 percent for attendants

**** Cost information could not be obtained.

However, there are currently no studies of smokers' willingness to pay for the right to smoke on aircraft. There could be losses in airline ridership due to smokers opting for other modes of transportation, but such losses could not be estimated in this study. In addition to partial smoking bans, options to curtail smoking also provide significant benefits at no apparent cost, particularly the option of a 10-minute smoking period every two hours. Such an option would, however, substantially raise short-term ETS levels and thereby increase acute health responses. For example, application of a steady-state model to the third flight (MD-80 with 25 smoking passengers) indicates that CO levels in the smoking section could be as high as 5 ppm if all passengers smoked during the 10-minute smoking period. The data from Cain et al. (1987) indicate that 10 percent of nonsmokers exposed to 5-ppm CO (due exclusively to tobacco smoking) for 10 minutes would express dissatisfaction due to eye irritation.

The other options listed in Table 9-9 either have costs that substantially exceed benefits (increased fresh-air intake) or very limited benefits (increased filter efficiency). Several manufacturers were contacted in an attempt to obtain estimates of filter costs, but the manufacturers were reluctant to divulge this information. Although the fuel penalty for increased fresh-air intake is quite small on a per-flight basis (\$10 to \$20), the aggregate costs are substantial. The fuel cost penalty was estimated from the relationship shown in Figure 9-6, which was derived from data provided in an NRC report (1986). The incremental fuel cost for a 50-percent increase in fresh-air intake ranges from \$0.04 per passenger-hour for DC-10-10 aircraft to \$0.067 for a B-727 aircraft. Multiplication by 769 million passenger-hours per year (see Table 9-8) yields an estimated cost range of \$30.8 to \$51.5 million for added fuel requirements.

9.3 APPLICATION OF FRAMEWORK TO POLLUTANTS

9.3.1 Cosmic Radiation

As noted earlier in this section, there are no practical approaches for reducing cosmic radiation levels on aircraft. Thus, the

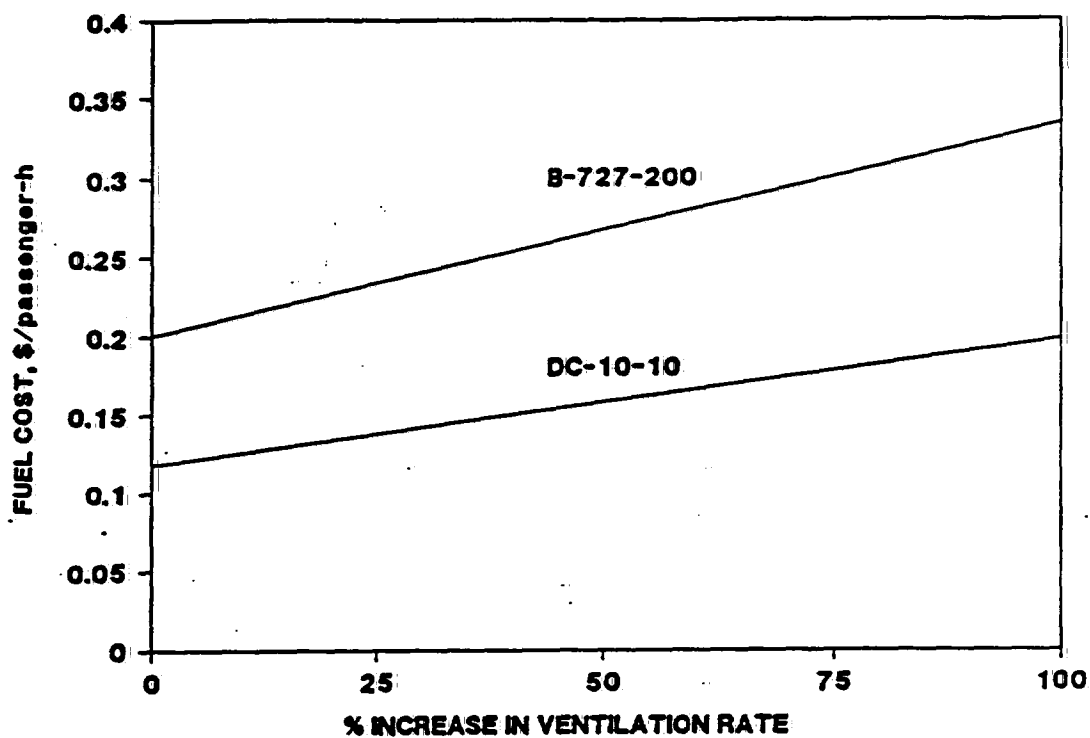


FIGURE 9-6. RELATIONSHIP BETWEEN INCREASE IN FRESH-AIR INTAKE AND FUEL COST PER PASSENGER-HOUR

only potential mitigation route involves the notion of exposure management. Through this strategy, excessive exposures could be reduced by avoiding extreme northern or southern latitudes and high altitudes where possible. Exposure management could also focus on specific types of personnel facing higher risks, such as female flight attendants in different stages of pregnancy, particularly the first trimester. This type of mitigation strategy applies equally to flight crew members, cabin crew members, and passengers.

9.3.2 Carbon Dioxide

Risk assessment was not performed for carbon dioxide (CO₂) because health effects of CO₂ exposure (other than those above occupational guidelines) have not been documented. Nonetheless, CO₂ levels exceeding 1,000 ppm, the level recommended by ASHRAE for satisfaction of comfort criteria, were measured on a substantial fraction of the monitored flights. Consequently, alternatives for reducing CO₂ levels in airline cabins were investigated but no cost-benefit analysis could be performed.

There are three types of options for reducing CO₂ levels--emissions reduction, increased ventilation, and removal by sorption. CO₂ removal could be achieved, for example, by passing air through an adsorbent bed mounted on a rotating drum or revolving belt (White 1989). Regeneration of the adsorbent would permit high capacity with low bed volume and weight. Continuous regeneration of the adsorbent would be accomplished by passing a small amount of purified air through a heated portion of the bed, then exhausting overboard the heated air containing high concentrations of CO₂. Aircraft waste heat from the lubrication oil system or engine exhaust gas would be used as the heat source for regeneration.

Emissions could be lowered by reduction of seating capacity, but this approach is not likely to be economically attractive to the airline industry. The potential effectiveness of remaining options, involving ventilation or removal, was investigated through modeling. Because the CO₂ sources (passengers and crew) are spread throughout the cabin, a

single-chamber model can be used. Using similar terminology to that used for the two-chamber model described earlier in this section (see Figure 9-2), the model for CO₂ in the cabin (C_{in}) can be stated as follows:

$$C_{in} = \frac{C_{out} \cdot MA + S}{(RA + L) - (1 - e) \cdot (SA - MA)}$$

The flights used previously for RSP modeling were also used for this modeling exercise. An emission rate of 0.3 l/min (18,000 ml/h) per passenger and an outdoor concentration of 330 ppm were assumed in making the calculations. The aircraft were assumed to be at full capacity--108 passengers for B-727 aircraft and 142 passengers for MD-80 aircraft.

CO₂ concentrations related to ventilation rates (currently measured levels and hypothetical increases up to 100 percent) are shown in Table 9-10. Concentrations are projected to decrease by about a third if the fresh-air intake rate were to be doubled. Thus, some flights with CO₂ levels above 1,000 ppm would likely remain under this scenario. As discussed earlier, this mitigation option would carry a fuel penalty and could also increase ozone levels and decrease humidity levels.

CO₂ concentrations related to filter removal efficiencies (zero assumed as current efficiency) are given in Table 9-11. (Discussions with a filter manufacturer indicated that removal efficiencies in the neighborhood of 50 to 75 percent may be attainable.) At a 50 percent removal efficiency, CO₂ levels could be reduced by 12 percent for current MD-80 aircraft (21 percent recirculation) or by 33 percent if the recirculation rate were as high as 50 percent).

9.4 REFERENCES

Cain, W.S., T. Tarik, L. See, and B. Leaderer. 1987. "Environmental Tobacco Smoke: Sensory Reactions of Occupants." Atmos. Environ. 21(2):347-353.

Fisher, A., D. Violette, and L. Chestnut. 1987. The Value of Reducing Risks of Death: A Note on New Evidence. Draft report, U.S. Environmental Protection Agency.

TABLE 9-10. PREDICTED CO₂ CONCENTRATIONS FOR THREE STUDY FLIGHTS
WITH HYPOTHETICAL INCREASES IN THE FRESH-AIR INTAKE RATE

Ventilation Rate	CO ₂ Concentration, ppm		
	Flight 1	Flight 2	Flight 3
Current level (base case)	873.2	1147.8	974.9
Increase of 25 percent	764.6 (12.4%)*	984.3 (14.2%)	845.9 (13.2%)
Increase of 50 percent	692.1 (20.7%)	875.2 (23.7%)	759.9 (22.1%)
Increase of 75 percent	640.4 (26.7%)	797.3 (30.5%)	698.5 (28.4%)
Increase of 100 percent	601.6 (31.1%)	738.9 (35.6%)	652.4 (33.1%)

* Numbers in parentheses indicate percent reduction in concentration from the base case.

TABLE 9-11. PREDICTED CO₂ CONCENTRATIONS FOR TWO STUDY FLIGHTS
WITH HYPOTHETICAL INCREASES IN FILTER EFFICIENCY

Filter Efficiency	CO ₂ Concentration, ppm		
	Flight 2 (21% recirc.)	Flight 3 (21% recirc.)	Flight 3 (50% recirc.)*
Zero (base case)	1147.8	974.9	1348.9
25 percent	1076.3 (6.2%)**	914.1 (6.2%)	1079.1 (20.0%)
50 percent	1013.2 (11.7%)	860.5 (11.7%)	899.3 (33.3%)
75 percent	957.0 (16.6%)	812.8 (16.6%)	770.8 (42.9%)

* Hypothetical recirculation rate for MD-80 aircraft.

** Numbers in parentheses indicate percent reduction in concentration from the base case.

Lorengo, D.G., and A. Porter. 1985. Aircraft Ventilation Systems Study. Final Report DTFA-03-84-C-0084. Atlantic City, NJ: U.S. Federal Aviation Administration Technical Center.

National Research Council. 1986. Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. National Academy Press. Washington, D.C.

Ryan, P.B., J. Spengler, and P. Halfpenny. 1988. "Sequential Box Models for Indoor Air Quality: Application to Airliner Cabin Air Quality". Atmos. Environ. 22(6):1031-1038.

U.S. Department of Transportation, Federal Aviation Administration. 1987. Airport Activity Statistics of Certificated Route Air Carriers: Twelve Months Ending December 31, 1987. U.S. Department of Transportation, Washington, D.C.

Viscusi, W.K. 1986. "The Valuation of Risks to Life and Health." In Bentkover et al., Benefit Assessment: The State of the Art. Dordrecht, Holland, Reidel Publishers.

White, D.H. 1989. "Modern Aircraft Cabin Air Purification Including Carbon Dioxide Removal." Pall Corporation, Glen Cove, NY.

Section 10.0
CONCLUSIONS AND RECOMMENDATIONS

2028397458

Section 10.0
CONCLUSIONS AND RECOMMENDATIONS

10.1 CONCLUSIONS

10.1.1 Measurement Methods and Results

The flights that were randomly chosen for monitoring in this study proved to be representative of the population of flights departing from major U.S. airports. Distributions of the monitored flights by airline and type of aircraft were very similar to those for all scheduled commercial jet aircraft flights.

Levels of particle-phase ETS contaminants monitored during the study were substantially higher in smoking sections of the aircraft than in nonsmoking areas. Respirable suspended particle (RSP) concentrations in the coach smoking section averaged about $175 \mu\text{g}/\text{m}^3$. The average RSP concentration in the no-smoking section near coach smoking (i.e., boundary region) was near $55 \mu\text{g}/\text{m}^3$, and RSP concentrations averaged about $35 \mu\text{g}/\text{m}^3$ in other no-smoking areas and on nonsmoking flights. These averages are based on combined results from two measurement methods--optical and gravimetric. One-minute peak RSP concentrations measured with optical sensors were more than ten times higher in the smoking section, and three times higher in the boundary region, than in the no-smoking areas on smoking flights. Measured RSP levels in the boundary region were most strongly correlated with observed smoking rates in the coach smoking section (i.e., higher levels when smoking rates were higher) and distance from the coach smoking section (i.e., higher levels at shorter distances).

Levels of gas-phase ETS contaminants that were monitored were also highest in smoking sections. Nicotine concentrations averaged near $13.5 \mu\text{g}/\text{m}^3$ in the coach smoking section, near $0.25 \mu\text{g}/\text{m}^3$ in the boundary region within the no-smoking section, and near or below $0.05 \mu\text{g}/\text{m}^3$ in other no-smoking areas and on nonsmoking flights. CO concentrations averaged near 1.4 ppm in the coach smoking section, near 0.7 ppm in no-smoking areas of smoking flights, and 0.6 ppm on nonsmoking flights.

Levels of these ETS tracers in the boundary region were not strongly correlated with observed smoking rates or distance from the coach smoking section.

Two separate techniques for estimating smoking rates on each monitored flight provided consistent results. Estimates based on technician observations of the number of lighted cigarettes during a one-minute interval every 15 minutes agreed well with estimates based on cigarette butts collected by technicians at the end of most smoking flights. An average of 20 cigarettes per hour, or 68 cigarettes per flight, was smoked by passengers in the coach smoking section on smoking flights that were monitored; an average of 13.7 percent of passengers were assigned to the coach smoking section.

Carbon dioxide (CO₂) levels on flights monitored during this study were frequently above the level recommended by ASHRAE (1,000 ppm) to satisfy comfort (odor) criteria. CO₂ concentrations on the monitored flights averaged above 1,500 ppm and exceeded 3,000 ppm on several occasions. Measured concentrations were 1,000 ppm or greater on 87 percent of the monitored flights, and the CO₂ levels were most strongly related to the number of passengers in the airliner cabin; on the average, 70 percent of the seats were occupied on the flights monitored in the study. Depending on assumed CO₂ exhalation rates, measured levels were as much as twice those predicted by a cabin air quality model. Even if the measured levels were to be lowered by half, however, CO₂ concentrations would still exceed 1,000 ppm on 24 percent of the study flights.

Relative humidity levels on monitored flights were quite low, averaging near 15 percent on smoking flights and near 20 percent on nonsmoking flights. Humidity levels were below 25 percent, outside the range indicated by ASHRAE for provision of adequate thermal comfort, on about 90 percent of all monitored flights. Temperatures in the cabins of monitored aircraft averaged near 24 °C (75 °F) for both smoking and nonsmoking flights and were within ASHRAE's comfort range.

Average levels of other pollutants (ozone, bacteria, and fungi) were relatively low on virtually all monitored flights. Measured levels of ozone did not exceed the FAA 3-hour standard of 0.1 ppm or the current EPA standard of 0.12 ppm on any of the monitored flights. The highest ozone level measured was 0.08 ppm, and the average measured level was between 0.01 and 0.02 ppm. Measured bacteria levels were somewhat higher in the smoking than no-smoking sections of monitored smoking flights, and the average level in the no-smoking section on these flights was nearly identical to that on nonsmoking flights. Measured fungi levels were somewhat higher on nonsmoking flights than smoking flights, but the bacteria and fungi levels in all cases were low, relative to those that have been measured in other environments.

The method used in the study to measure air exchange rates was generally adequate for aircraft with recirculation but was inadequate for other types of aircraft. The measurement method, involving release and sampling of perfluorocarbon tracers, was less effective on aircraft without recirculation because of the limited extent of lateral air movement on such aircraft. This limitation could have been overcome by increasing the number of tracer release and sampling locations, but such a strategy was deliberately avoided in this study in order to remain unobtrusive to passengers and flight attendants during monitoring.

The strategy of monitoring at multiple seat locations provided important insights regarding spatial variations in cabin air quality, particularly for ETS contaminants. This strategy provided some indications that the boundary region in the no-smoking section was affected by coach smoking, in addition to the distinct effects in the smoking section itself, and that spatial variations were relatively minor for CO₂ and other pollutants (ozone, bacteria, and fungi) that were monitored.

The strategy of continuous monitoring where practical, combined with integrated sampling, also provided some important insights concerning cabin air quality. Continuous monitoring results provided the strongest indication of an effect of smoking in the no-smoking boundary region.

10.1.2 Risk Assessment

The risks faced by cabin crew members and passengers depend on such factors as frequency of flying, number of years flown, specific routes flown, and, in the case of ETS exposures, seat locations and prevailing smoking rates. The study conclusions pertaining to cancer risks are based on specific scenarios relating to number of hours per year in flight, number of years flown, and, in the case of ETS exposures, proportion of time spent in the smoking section, boundary region near smoking, and other no-smoking areas. Detailed descriptions of the scenarios and calculations underlying the risk estimates given herein are provided in Section 7.0 for ETS and in Section 8.0 for cosmic radiation. Estimates for cabin crew members relating to ETS exposure pertain only to flight attendants and do not include the cockpit crew.

ETS

Estimated lifetime lung cancer risks ascribable to ETS exposure for nonsmoking cabin crew members flying 960 hours per year on smoking flights for 20 years range from 12 to 15 premature cancer deaths per 100,000 nonsmoking cabin crew members for domestic flights and from 13 to 17 premature cancer deaths per 100,000 for international flights. The range of estimates was derived from two different cancer risk models (a phenomenological model and a multistage model) that assume different durations of exposure. Applying these risk estimates to the entire U.S. cabin crew population results in an estimated 0.18 premature lung cancer deaths per year for domestic flights (that is, approximately 4 premature deaths can be expected every 20 years) and 0.16 premature deaths per year for international flights.

Estimated lifetime lung cancer risks due to ETS exposure for nonsmoking passengers flying 480 hours per year on smoking flights for

30 years range from 0.3 to 0.8 premature cancer deaths per 100,000 nonsmoking passengers for domestic flights and from 0.2 to 0.6 premature cancer deaths per 100,000 for international flights. The range of estimates was derived from the two cancer risk models mentioned above, and the relatively broad range is due to differences in assumed durations of exposure and the sensitivity of the multistage model to assumptions concerning the age at which exposure begins.

Estimated lifetime lung cancer risks due to ETS exposure for nonsmoking passengers flying 48 hours per year on smoking flights for 40 years are approximately 0.1 premature cancer deaths per 100,000 for both domestic and international flights. Applying these risk estimates to the U.S. flying population results in an estimated 0.24 premature lung cancer deaths per year for domestic flights (that is, approximately 10 premature deaths can be expected every 40 years) and 0.12 premature deaths per year for international flights.

In terms of acute effects based on CO concentrations as a proxy for ETS levels, it is estimated that on one-third of smoking flights about 1 in 8 persons seated in the smoking section would experience irritation due to ETS exposure. Further, it is estimated that on about one-third of domestic smoking flights, ETS levels in the smoking section (based on nicotine concentrations as a proxy) would be sufficiently high to evoke a marked sensory response in the eye and nose of an airliner cabin occupant.

Differential effects of ETS and its constituents on such sensitive populations as asthmatics, children, and persons with ischemic heart disease or other cardiovascular disease could not be estimated.

Cosmic Radiation

Estimated lifetime cancer risks due to cosmic radiation exposure for cabin crew members flying 960 hours per year range from 5 to 61 premature deaths per 100,000 individuals flying for 20 years on domestic flights and from 13 to 30 premature deaths per 100,000 individuals flying for 10 years on international flights. The estimates, which

pertain to cockpit crew members as well as cabin crew members, are lowest for relatively short north-south domestic flights and higher for coast-to-coast flights involving higher altitudes. The highest estimates are for relatively long, circumpolar international flights which also occur at high altitudes.

Estimated lifetime cancer risks due to cosmic radiation exposure for passengers flying 480 hours per year range from 3 to 30 premature deaths per 100,000 individuals flying for 20 years on domestic flights and from 7 to 15 premature deaths per 100,000 individuals flying for 10 years on international flights. Like the above estimates for cabin crew, the range is governed largely by flight altitudes and latitudes. Another concern is the effect of cosmic radiation on a fetus, particularly during the first trimester.

Other Pollutants

The levels of bacteria and fungi measured in the airliner cabin air in this study were found to be below the levels generally thought to pose risk of illness. Because quantitative dose-response information on the health risks of biological aerosols was not available, the evaluation of the concentration data was performed by placing the prevalence of individual genera that were identified in rank order, and comparing the prevalence to biological aerosols in other indoor environments. The levels and genera measured in the cabin environment were similar to or lower than those commonly encountered in indoor environments characterized as "normal."

It was unnecessary to perform a risk assessment for ozone because measured levels on all monitored flights were well below the current FAA and EPA standards.

10.1.3 Mitigation

Among the methods evaluated for reducing risks due to ETS, a total ban on airliner cabin smoking would eliminate ETS exposure in airliner cabins and yield the greatest benefit to flight attendants

and nonsmoking passengers. A total ban on smoking on domestic flights is estimated to result in an annual benefit of approximately \$3 million to cabin crew and passengers, based on reduced mortality risks. In conducting this benefit/cost analysis, reduction in mortality and associated economic benefits were considered but benefits relating to reduced morbidity were not. Possible costs related to smokers' inconvenience and discomfort or to displacement of smokers to other modes of transportation were not considered due to limited data.

Beyond the two-hour ban that reduces ETS exposures on domestic flights by approximately 45 percent, more restrictive bans could be implemented to reduce exposures by as much as 98 percent. Restricting smoking to flights of a 6-hour or greater duration would reduce ETS exposures by approximately 98 percent, and a restriction for flights of 4 hours or longer would reduce exposures by about 86 percent. A different type of strategy to curtail smoking, such as allowing smoking for a 10-minute period every two hours, could reduce average exposures to ETS by as much as 70 percent. Such a strategy, however, could substantially increase the risks of health effects from acute exposure during the brief periods when smoking would be allowed.

Two other mitigation measures--increased ventilation and improved filter efficiency--would reduce ETS exposures by lesser amounts, ranging from 5 to 33 percent. Annual costs of increased ventilation (\$6 to \$50 million), which could reduce ETS exposures by as much as 33 percent, are substantially higher than the benefits (\$0.7 to \$1.0 million) that could be calculated within the constraints of this study. Costs related to improved filter efficiency were not available, but improved efficiency would provide only a marginal reduction (5 percent) in ETS exposures.

Exposure management is the only viable option for reducing cabin crew member and passenger exposures to cosmic radiation. In the case of crew members, this strategy would involve careful scheduling of personnel to avoid persistent exposure to higher cosmic radiation levels generally associated with high-altitude flights and flight paths toward extreme northern or southern latitudes.

On aircraft with recirculation, CO₂ could be removed by sorption on solid adsorbent beds whose adsorbent capacity for CO₂ can be regenerated by heating. Increased ventilation could also bring CO₂ levels closer to the guidelines specified by ASHRAE. Cost or reliability data for a sorption system were not available for comparison with costs of additional ventilation.

In view of the low levels observed for ozone and biological aerosols, mitigation strategies were not assessed for these pollutants.

10.2 RECOMMENDATIONS

10.2.1 Actions for Improving Cabin Air Quality

Consideration should be given to a total ban on smoking on all flights departing from or arriving at U.S. airports as a means of eliminating the ETS risks currently faced by nonsmoking passengers and nonsmoking cabin crew members. The estimated benefits of such a strategy exceed the costs, based on currently available data. In considering this ban, consideration will need to be given to smokers' inconvenience and discomfort, possible economic consequences of displacement of smokers to alternative transportation modes, and other potential consequences such as smoker withdrawal symptoms. Possible alternatives include limiting smoking to longer-duration flights or restricting the time periods when smoking is allowed on flights. In the latter case, further study would be needed of the potential health effects from acute exposure that could occur during the limited periods when smoking would be allowed.

Airlines should implement exposure-management strategies to reduce risks faced by cabin crew members, particularly those related to cosmic radiation. Such strategies would include careful scheduling of personnel, especially those at highest risk, to avoid persistent higher exposures associated with flight paths at extreme northern/southern latitudes and higher altitudes.

Sorption should be considered as a means of reducing CO₂ levels in airliner cabins. The feasibility of implementing this approach needs

to be further explored, along with potential costs, benefits, and practical considerations. Such an approach, or increased ventilation, could also reduce levels of other potentially hazardous chemicals, such as volatile organic compounds that were not measured during this study.

No actions need to be taken to reduce currently prevailing levels of ozone or biological aerosols. The types of preventive strategies that are currently in place for ozone, which may be partly responsible for the relatively low levels measured during this study, should be continued.

10.2.2 Information Needs

Due to constraints of unannounced and unobtrusive monitoring required to meet study objectives, this study could not take full advantage of the currently available state-of-the-art instrumentation for pollutant monitoring. Based on observations and conclusions from this study, the following areas of further study are recommended:

Additional measurements of CO₂ should be performed in commercial airliner cabins. Such measurements need to be conducted with continuous monitoring devices on different types of aircraft and at different levels of passenger occupancy.

A study of flight attendants' exposures with personal monitors should be conducted if a total ban on smoking is not enacted. Due to study limitations, flight attendants' exposures could not be estimated directly. A personal monitoring study of flight attendants would improve estimates of exposures by accounting for the different breathing height from that of passengers and time spent in areas such as galleys, which were not monitored during this study.

Further measurements of prevailing air exchange rates on aircraft should be performed. Due to the need to remain unobtrusive during this study, it was not possible to widely deploy sources and samplers to obtain more reliable measurements. Improved estimates will provide a stronger basis for cabin air quality modeling which is crucial to assessment of mitigation strategies related to ventilation.

Further information on special populations and short-term health effects would support improved risk assessments. The information required includes (1) the flying frequency of children and sensitive individuals such as asthmatics, (2) dose-response functions relating various types of short-term health effects (e.g., eye/nose/throat irritation) to levels of various ETS tracers, and (3) quantitative measures of ETS effects on the cardiovascular system of individuals with pre-existing cardiovascular disease.

APPENDIX A

**EXPLANATION AND SENSITIVITY ANALYSIS OF
THE MODIFIED ARMITAGE AND DOLL MODEL (LesLife®)**

2028397469

Section 1.0

EXPLANATION OF THE MODIFIED ARMITAGE AND DOLL MODEL

1.1 General Approach

The approach to risk assessment involves converting the ambient air data to a risk-equivalent dose. The underlying assumptions in the Modified Armitage and Doll Model, which is a modification to the original model presented by Armitage and Doll (1961), are that:

- RSP is a reliable indicator for estimating the relationship between exposure to cigarette smoke and health risks.
- Risk data exist for wives of smoking husbands; their relative risk is approximately 1.3 based on case-control studies.
- Spousal exposure can be inferred from measurements made of the impact of an individual smoker on indoor air quality together with empirical statistics on the duration of marriages.
- For the multistage model of carcinogenesis with five stages, the following question can be posed:

If X years of exposure at level Y causes a relative risk of 1.3, what is the dose-response coefficient?

- The dose response coefficient generated in the step above is then used, along with a five-stage multistage model and dose estimates derived from aircraft monitoring data, to calculate risks to the selected populations of interest.

1.2 Specific Application

The central problem in risk assessment is scaling a risk estimate that is valid for lifetime exposure in a form that is useable in models designed for less-than-lifetime exposure. The multistage theory of carcinogenesis can assist in providing a solution by considering age-specific risks of exposure to a fixed concentration of a carcinogen for some fixed duration. If either the first stage (tumor initiation) or the penultimate fourth stage (tumor promotion) is affected, the age-specific cancer rate (hazard), $R(t)$, for exposure from birth is given by (Day and Brown 1980; Brown and Chu 1982):

$$R(t) = cE(t-\delta)^{K-1}, \quad t > \delta \quad (1)$$

where

- $R(t)$ = the age specific cancer rate
- t = attained age,
- E = exposure or dose level
- c = a dose response coefficient which converts E to risk
- δ = the latency period required to convert an initiated tumor into a fatal cancer
- K = the number of stages assumed to be acting in the carcinogenesis process.

Day and Brown (1982) also present formulae relating rate at age, t , to some less-than-lifetime exposure. Assuming an exposure of duration, D , starting at age, t_0 , and ending at time, t_1 , age-specific risk can be predicted. Risk is zero for the period, t_0 to $t_0+\delta$, because this is the definition of latency. For an initiator:

$$R(t) = cE(t-t_0-\delta)^{K-1}, \quad t_1+\delta \geq t > \delta \quad (2)$$

and

$$R(t) = cE[(t-t_0-\delta)^{K-1} - (t-t_1-\delta)^{K-1}], \quad t > t_1+\delta \quad (3)$$

For low risks, lifetime risk, L is given approximately by:

$$L \approx \sum_{t=0}^{100} S(t) * 0.5[R(t) + R(t+1)] \quad (4)$$

where

- $S(t)$ = the probability of surviving from birth to age t in some standard population.

For (1) this is

$$L \approx \sum_{t=6}^{100} S(t) * cE * 0.5 * [(t-6)^{K-1} + (t+1-6)^{K-1}] \quad (5)$$

A similar, but more complicated, expression can be derived from (2) and (3):

$$L \approx cE * 0.5 * \left[\sum_{t=t_0+6}^{t_1+6} S'(t) * [(t-\tau)^{K-1} + (t+1-\tau)^{K-1}] + \sum_{t_1+6+1}^{100} S'(t) * [(t-\tau)^{K-1} + (t+1-\tau)^{K-1} - (t-\Omega)^{K-1} - (t+1-\Omega)^{K-1}] \right] \quad (6)$$

Here

$$\tau = t_0 + 6$$

$$\Omega = t_1 + 6$$

$S'(t)$ = the probability of survival from t_0 to some age t .

Equation 6 can now be rearranged to yield an estimate of c , the dose-response coefficient:

$$c = \frac{2 \times L}{\#} \quad (7)$$

Here

$\#$ = the term in large brackets of equation (6).

Assuming values for K , δ , and some survival function $S(t)$, we have scaled a unit cancer risk (risk coefficient, q^*) to apply to less than lifetime, or time-varying exposure. Since reasonable guesses can be made about K (4 or 5) and δ (5 to 10 years), and since survival functions for recent U.S. populations have not varied dramatically, this procedure is defensible. [The exact survival functions used here are derived from the 1980 U.S. Decennial lifetables (National Center for Health Statistics, 1985).] Nevertheless, estimates of several important parameters, L , E , t_0 , and t_1 must be derived from ancillary information.

In the case of L, studies of lung cancer risk in nonsmoking women married to men who are cigarette smokers suggest that their relative risk is about 1.3 relative to nonsmoking women married to nonsmokers (National Research Council 1986; Blot and Fraumeni 1986), and studies of nonsmokers have shown that the lifetime risk of lung cancer in nonsmoking women is about 630 deaths per 100,000 women at risk (Garfinkel 1981; Ginevan and Mills 1986). Taken together these two values suggest that the lifetime risk attributable to spousal exposure to ETS is about 630 deaths per 100,000 women at risk $\times 0.30$, or 189 deaths per 100,000.

This value is slightly overestimated because some nonsmoking women in the cohort used to generate the overall lung cancer risk of 630 deaths per 100,000 are married to smokers. If 30 percent of these women are assumed, in fact, to be married to smokers, and that those married to smokers have a relative risk of 1.30, then the risk in a nonsmoking woman married to a nonsmoker can be calculated and used as the basis of the attributable risk calculation in this investigation. If the proportion of women married to smokers were as high as 0.30 [some of the women were unmarried, and nonsmoking women tend to marry nonsmoking men (National Research Council 1986)], then the overall relative risk in the population would be $(0.7 \times 1.0) + (0.3 \times 1.3) = 1.09$. If the overall absolute risk in the population, 630/100,000, is divided by the overall relative risk, 1.09, a baseline absolute risk of 578 per 100,000 occurs in nonsmoking women married to nonsmokers. Thus, the adjusted attributable risk is 0.3×578 or 174 per 100,000. The following calculations will take into account this adjusted attributable risk for the value of L.

Having defined a risk coefficient, an exposure term, E, must also be determined. Spengler (1981) has shown that residences with one smoker average $12.4 \mu\text{g}/\text{m}^3$ RSP, and residences with two smokers average $46.3 \mu\text{g}/\text{m}^3$ more RSP than those with no smokers. In a separate study Spengler (1985) found that residences with smokers averaged $46 \mu\text{g}/\text{m}^3$ RSP higher than those who do not reside with smokers.

A computational approach can be taken to determine residential RSP levels attributable to smoking. If the average nonsmoking residence is

2
26 mg/day
⇒ 150 µg/m³

assumed to experience 0.8 air exchanges and has an effective volume of 340 m³, smoking 1 cigarette per hour in that dwelling will add approximately 150 µg/m³ to the RSP levels in the home. However, this calculation assumes that a cigarette is smoked every hour. In fact, working men and women spend approximately 6 waking hours per day at home (Repace and Lowrey, 1985). Therefore, to obtain the actual average, the 150 µg/m³ must be divided by 4, which yields 37.5 µg/m³. This is in good agreement with the values Spengler reported earlier, providing a basis for assuming that spousal smoking exposure corresponds to an increment of approximately 40 µg/m³ RSP exposure. The uncertainty in this value is probably within a factor of two (e.g., the true average exposure could be as little as 20 µg/m³ RSP or as much as 80 µg/m³ due to spousal smoking).

An estimate of the average respiratory rate and the duration of daily exposure for a woman exposed to a husband who smokes is necessary to complete our exposure estimate. For this calculation, it is assumed that a woman in the home spends 1/3 of the time resting, and the other 2/3 engaged in alternate sitting and light work. This scenario implies a respiratory rate of about 0.8 m³ per hour. Activity surveys show that working women spend 15 hours per day in the home, and that nonworking women spend more than 20 hours per day in the home. However, some of the time spent outside the home would still be spent in the presence of the smoker spouse, and for the case of a working woman, time spent within the home would likely occur when the spouse was also present. These considerations lead to the adoption of a 24-hour/day regime as a reasonable exposure scenario. Thus, a woman married to a smoker might be expected to receive a daily dose, D, of:

$$\begin{aligned} D &= 0.8 \text{ m}^3/\text{hour} \times 24 \text{ hours/day} \times 40 \text{ } \mu\text{g}/\text{m}^3 \\ &= 0.77 \text{ mg/day} \end{aligned} \quad (8).$$

For duration of exposure, if a woman is assumed to become married at age 22 to a man aged 25, the exposure will likely terminate at the husband's death which, for a smoker, will occur at approximately age 65. This implies an exposure duration of 40 years. However, several of the

spousal smoking studies were conducted in countries where, because of custom and culture, the husband may be ten or more years older than the wife at marriage. This consideration, together with factors such as second marriages and separations without divorce, suggest that this duration should be revised downward. Duration of exposure is therefore taken to be 35 years beginning at age 22, recognizing that this duration could be 5 years higher or lower.

One can easily verify that, taken together, the assumptions developed here imply a phenomenological risk estimate very similar to that developed by Repace and Lowrey (1985). That is, assuming a daily exposure of 0.77 mg results in a lifetime cancer risk of 174 per 100,000 persons. Since each person is at risk for about 35 years, this implies an overall risk, Z, of:

$$\begin{aligned} Z &= \frac{174 \text{ deaths}}{3.5 \text{ million person-years} \times 0.77 \text{ mg/day}} \\ &= 6.45 \text{ deaths per } 100,000 \text{ person year/mg/day} \quad (9), \end{aligned}$$

only 30 percent greater than the risk estimated by Repace and Lowrey.

The necessary inputs are thus provided in order to parameterize the model given in equation (7):

L = 174 deaths per 100,000 or 0.00174

E = 0.77 mg/day

t₀ = 22

t₁ = 57.

The model will be parameterized assuming

δ = 5 or 10, and

k = 4 or 5.

The results of this parameterization are presented for a scenario in which exposure starts at age 25 and continues for 25 years; this can reasonably be applied to a cabin crew member (Table A-1). All entries in the table are in units of risk per 100,000 persons [lifetime], assuming a

average daily exposure of 1 mg per day. To obtain the risk of exposure at different levels, the actual daily average is multiplied by the table entry for a given model.

TABLE A-1. RISK COEFFICIENTS

Model	Risk per 100,000 [lifetime]
k=4, δ =5	170
k=4, δ =10	170
k=5, δ =5	167
k=5, δ =10	165

For this scenario, the exact model parameterization is not important. If one applies a risk factor of 170 per 100,000 to the 0.5 mg/day RSP inhalation calculated earlier for a female flight attendant, a lifetime risk of 170×0.5 or 85 per 100,000 results, a factor of approximately three higher than the estimate obtained by using the Repace and Lowrey model.

Section 2.0

SENSITIVITY OF THE MODIFIED ARMITAGE AND DOLL MODEL

The preceding discussion demonstrates that cancer risk estimates developed using the Modified Armitage and Doll Model (based on multistage theory and data on spousal smoking studies) do not differ greatly from cancer risk estimates developed using the Phenomenological Model (based on data on lung cancer incidence in Seventh Day Adventists). However, it is still of interest to determine how differing assumptions regarding the actual choice of model parameters might affect risk estimates. For this reason, the analyses presented in Figure A-1 were performed to demonstrate that alteration of model assumptions does not significantly alter the outcome. The calculations providing the results in Figure A-1 were based on two scenarios representing extreme circumstances: exposures commencing at age 5 or age 30, and continuing for 40 years. Each scenario was examined assuming three different multistage models. In two models, ETS was assumed to be an initiator; in one of these two models, the number of stages (K) was taken to be 4 and latency (δ) was taken as 5 years; in the other of these two models, K was taken as 5 and δ was taken as 10. In the third model, ETS was assumed to be a penultimate stage carcinogen (promoter); K was taken as 5, and δ was taken as 10.

As illustrated by Day and Brown (1980), initiators and promoters can have very different implications for cancer risk, especially when the age at first exposure varies dramatically. This observation is borne out for the case of exposure starting at age 5 if ETS is assumed to be a promoter. Here, if the duration of exposure is short, risk is markedly lower than for the other models. Similarly, the highest risk for an initiator is attained by assuming a five-stage model in which exposure commences in childhood. However, for exposure in an adult, risk varies relatively little with changes in assumptions. The reason for this invariance is that, if one accepts the spousal smoking studies as valid, assessing risk to airliner passengers and cabin crew members involves little extrapolation. If risks in the wives of smoking husbands are really known, expo-

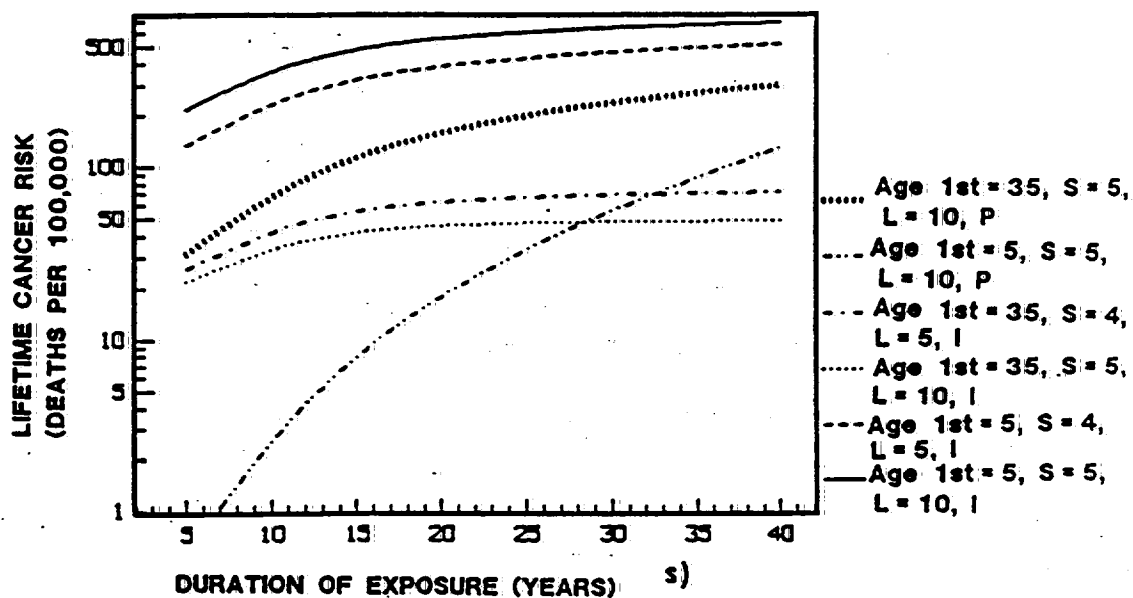


FIGURE A-1. CANCER RISKS USING FOUR- AND FIVE-STAGE RISK MODELS. S=NUMBER OF STAGES; L=LATENCY OF 5 OR 10 YEARS; I=ETS INITIATOR; P=ETS PROMOTER; AGE=AGE AT FIRST EXPOSURE

asures aboard airliners are not so different that any reasonable modeling approach would be expected to give greatly different risks. It follows that the approach of the Phenomenological Model involves little extrapolation and presents similar results to the Modified Armitage and Doll Model because the risks estimated from spousal smoking studies and the risks estimated from Seventh Day Adventists are similar.

As a further check on model robustness, the spousal smoking data used to parameterize the Modified Armitage and Doll Model and the Seventh Day Adventist data in the Phenomenological Model were used to estimate the c parameter given in equations 2 through 7 in Section 1 of this appendix. The model parameterizations are summarized in Table A-2. While the model parameterizations differ, the c values are identical. In risk terms, this means that regardless of the data used to parameterize the Modified Armitage and Doll Model or the particular risk scenario considered, the risks are the same. This exercise further demonstrates that the small differences seen between the Phenomenological and Modified Armitage and Doll Models are attributable to the models themselves, not to the underlying data.

It is difficult to identify other factors that would greatly affect the risk estimates provided by the Modified Armitage and Doll Model. One could postulate some pervasive, but unknown, source of bias in spousal smoking studies which generates apparent effects where they do not in fact exist; or one could also suggest other factors which give a bias of similar magnitude in Seventh Day Adventists. However, such explanations are hardly warranted. One could also suggest that the exposures assumed are much too low (and thus inflate risk), but in both the Phenomenological and the Modified Armitage and Doll Models, the assumed exposures are more likely to be slightly too high than much too low and are, in any case, supported by the available literature (National Research Council 1986). For these reasons, the risks as calculated are unlikely to be high or low by more than a factor of three, and are more likely to understate risk slightly than they are to overstate risk greatly.

TABLE A-2. MODEL PARAMETERIZATIONS USED IN THE ANALYSES
OF MODEL ROBUSTNESS

	Seventh Day Adventist Data Used in the Phenomenological Model	Spousal Smoking Data Used in the Modified Armitage and Doll Model
Deaths per 100,000	300	174
Age at first exposure	20	22
Duration (years)	45	35
Intake (mg/day)	1.43	0.77
Multistage coefficient (c)	2.68×10^{-11}	2.76×10^{-11}

Section 3.0

REFERENCES

- Armitage, P. and R. Doll. 1961. "Stochastic models for carcinogenesis." Proceedings of the fourth Berkeley Symposium on Mathematical Statistics and Probability. Volume 4:19-38. University of California Press, Berkeley, California.
- Blot, W.J. and J.F. Fraumeni. 1986. "Passive smoking and lung cancer." J. Nat. Cancer Inst. 77:993-1000.
- Brown, C.C. and K.C. Chu. 1982. "Approaches to epidemiologic analysis of prospective and retrospective studies: Example of lung cancer and exposure to arsenic." In: Environmental Epidemiology: Risk Assessment. R.L. Prentice and A.S. Whittemore eds. SIAM, Philadelphia, pp 94-106.
- Day, N.E. and C.C. Brown. 1980. "Multistage models and primary prevention of cancer." J. Nat. Cancer Inst. 64: 977-989.
- Garfinkel, L. 1981. "Time trends in mortality among nonsmokers and a note on passive smoking." J. Nat. Cancer Inst. 66:1061-1066.
- Ginevan, M.E. and W.A. Mills. 1986. "Assessing the risks of radon exposure: The influence of cigarette smoking." Health Physics 51:163-174.
- National Center for Health Statistics. 1985. U.S. Decennial Lifetables for 1979-81. DHHS Publication No. (PHS) 85-1150-1. U.S. Government Printing Office, Washington, DC.
- National Research Council. 1986. Environmental Tobacco Smoke: Measuring Exposures and Assessing Health Effects. National Academy Press. Washington, D.C.
- Repace, J.L. and A.H. Lowrey. 1985. "A quantitative estimate of nonsmokers' lung cancer risk from passive smoking." Environ. International 11:3-22
- Spengler, J.D., D.W. Dockery, W.A. Turner, J.M. Wolfson, and B.J. Ferris Jr. 1981. "Long-Term measurements of respirable sulfates and particulates inside and outside homes." Atmos. Environ. 15:23-30
- Spengler, J.D., R.D. Treitman, T.D. Tosteson, D.T. Mage, and M.L. Soczek. 1985. "Personal exposures to respirable particulates and implications for air pollution epidemiology." Environ. Sci. Technol. 19:700-707.

APPENDIX B

**INCREMENTAL RISKS OF PREMATURE LUNG CANCER DEATH AMONG
NONSMOKERS FROM EXPOSURE TO RESPIRABLE SUSPENDED PARTICULATE,
ASCRIPTABLE TO ENVIRONMENTAL TOBACCO SMOKE ON SMOKING FLIGHTS**

2028397482

TABLE B-1. INCREMENTAL RISK OF PREMATURE LUNG CANCER DEATH AMONG
NONSMOKING CABIN CREW MEMBERS FROM EXPOSURE TO RSP,
ASCRIPTABLE TO ETS ON SMOKING FLIGHTS.
RISKS ARE BASED ON THE MODIFIED ARMITAGE AND DOLL MODEL.

		Risk in Lifetime Deaths per 100,000 Exposed		
Age at Start	Years Flown	Hours Flown per Year		
		300	600	900
<u>Domestic Flights</u>				
15	10	7.7555	15.511	23.267
15	20	11.313	22.626	33.939
15	30	12.708	25.417	38.125
15	40	13.146	26.293	39.439
25	10	3.5574	7.1148	10.672
25	20	4.9528	9.9055	14.858
25	30	5.3908	10.782	16.173
25	40	5.4894	10.979	16.468
35	10	1.3954	2.7907	4.1861
35	20	1.8334	3.6669	5.5003
35	30	1.9319	3.8639	5.7958
35	40	1.9449	3.8898	5.8347
<u>International Flights</u>				
15	10	8.6595	17.319	25.979
15	20	12.632	25.263	37.895
15	30	14.190	28.379	42.569
15	40	14.679	29.358	44.036
25	10	3.9721	7.9441	11.916
25	20	5.5301	11.060	16.590
25	30	6.0192	12.038	18.058
25	40	6.1292	12.258	18.388
35	10	1.5580	3.1160	4.6740
35	20	2.0472	4.0943	6.1415
35	30	2.1571	4.3143	6.4714
35	40	2.1716	4.3432	6.5148

TABLE B-2. INCREMENTAL RISK OF PREMATURE LUNG CANCER DEATH AMONG NONSMOKING PASSENGERS FROM EXPOSURE TO RSP, ASCRIBABLE TO ETS ON SMOKING FLIGHTS.
RISKS ARE BASED ON THE MODIFIED ARMITAGE AND DOLL MODEL.

		Risk in Lifetime Deaths Per 100,000 Exposed			
Age at Start	Years Flown	Hours Flown Per Year			
		45	100	450	900
<u>Domestic Flights</u>					
5	10	0.2081	0.4624	2.0808	4.1617
5	20	0.3151	0.7003	3.1514	6.3028
5	30	0.3642	0.8094	3.6424	7.2849
5	40	0.3835	0.8522	3.8350	7.6701
15	10	0.1071	0.2379	1.0705	2.1411
15	20	0.1562	0.3470	1.5616	3.1232
15	30	0.1754	0.3898	1.7542	3.5084
15	40	0.1815	0.4033	1.8147	3.6294
25	10	0.0491	0.1091	0.4911	0.9821
25	20	0.0684	0.1519	0.6837	1.3673
25	30	0.0744	0.1654	0.7441	1.4883
25	40	0.0758	0.1684	0.7577	1.5155
35	10	0.0193	0.0428	0.1926	0.3852
35	20	0.0253	0.0562	0.2531	0.5062
35	30	0.0267	0.0593	0.2667	0.5334
35	40	0.0268	0.0597	0.2685	0.5369
<u>International Flights</u>					
5	10	0.1526	0.3391	1.5259	3.0519
5	20	0.2311	0.5136	2.3110	4.6220
5	30	0.2671	0.5936	2.6711	5.3422
5	40	0.2812	0.6250	2.6124	5.6247
15	10	0.0785	0.1745	0.7851	1.5701
15	20	0.1145	0.2545	1.1452	2.2904
15	30	0.1286	0.2859	1.2864	2.5728
15	40	0.1331	0.2957	1.3308	2.6615
25	10	0.0360	0.0800	0.3601	0.7202
25	20	0.0501	0.1114	0.5014	1.0027
25	30	0.0546	0.1213	0.5457	1.0914
25	40	0.0556	0.1235	0.5557	1.1113
35	10	0.0141	0.0314	0.1412	0.2825
35	20	0.0186	0.0412	0.1856	0.3712
35	30	0.0196	0.0435	0.1956	0.3911
35	40	0.0197	0.0438	0.1969	0.3938

TABLE B-3. INCREMENTAL RISK OF PREMATURE LUNG CANCER DEATH AMONG NONSMOKERS FROM EXPOSURE TO RSP, ASCRIBABLE TO ETS ON SMOKING FLIGHTS. RISKS ARE BASED ON THE PHENOMENOLOGICAL MODEL.

Risk in Lifetime Deaths per 100,000 Exposed				
Years Flown	Cabin Crew Members Hours Flown Per Year			
	300	600	900	
<u>Domestic Flights</u>				
10	2.0096	4.0192	6.0288	
20	4.0192	8.0384	12.058	
30	6.0300	12.060	18.090	
40	8.0400	16.080	24.120	
<u>International Flights</u>				
10	2.2438	4.4877	6.7315	
20	4.4877	8.9753	13.460	
30	6.7320	13.464	20.196	
40	8.9760	17.952	26.928	
<u>Passengers</u>				
	Hours Flown Per Year			
	45	100	450	900
<u>Domestic Flights</u>				
10	0.0277	0.0616	0.2774	0.5548
20	0.0555	0.1233	0.5548	1.1096
30	0.0832	0.1850	0.8323	1.6645
40	0.1110	0.2466	1.1097	2.2194
<u>International Flights</u>				
10	0.0203	0.0452	0.2034	0.4068
20	0.0407	0.0904	0.4068	0.8137
30	0.0610	0.1356	0.6103	1.2205
40	0.0814	0.1808	0.8137	1.6274